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INDUSTRIAL MEDICINE

The Journal of Occupational Diseases and Traumatic Surgery

April, 1941

The Pittsburgh Meeting
— Institute of Industrial Medicine and Industrial Hygiene, May 5-9 —

Sulfathiazole
— Present Status in the Treatment of Infections —

Types of Goitre
— Characteristics, and Results of Treatment —

Head Lesions
— Evaluation —

The Nurse in Industry
— Responsibility to the Physician —

Industrial Poisonings
— Practical Aspects —

Dermatitis
— Prevention in Industry —

A Health and Medical Plan
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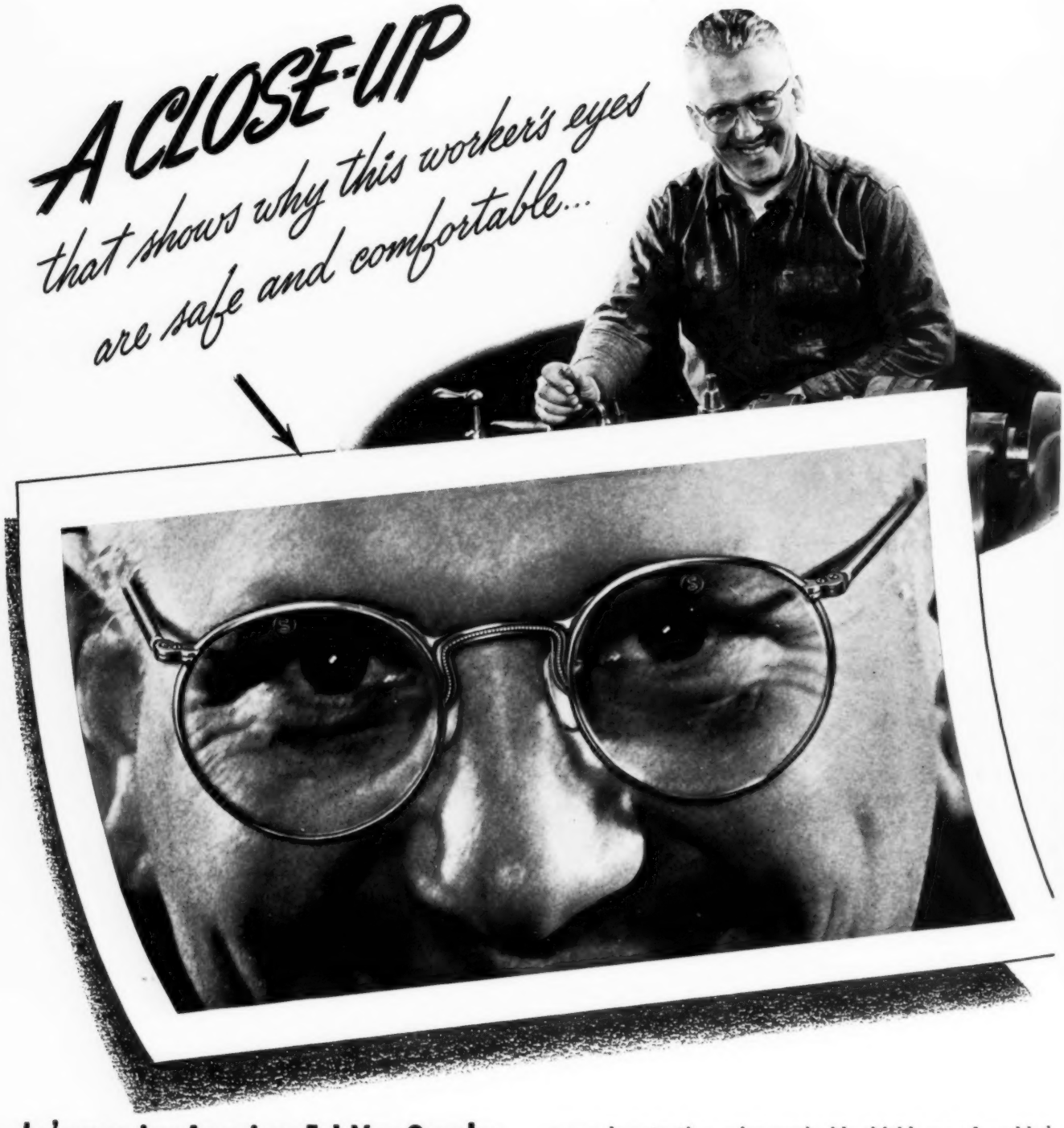

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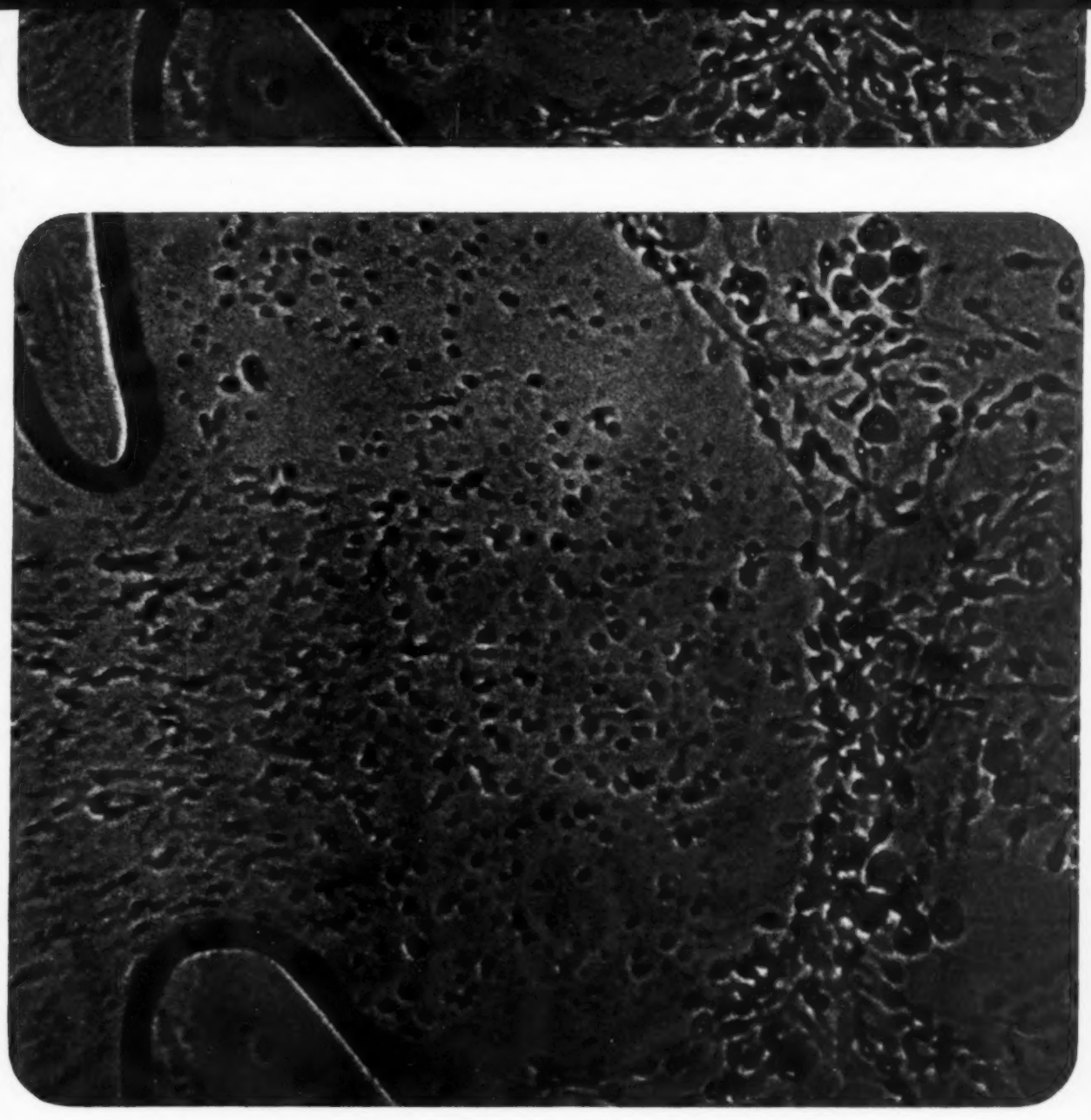

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The Toxicity of Antimony
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DR. RAMMELKAMP'S report on the present status of "Sulfathiazole" in the treatment of infections, which is the opening article this month, is an important contribution to the rapidly growing literature of a new subject. It is so well conceived and studiously executed as to command a permanent usefulness, regardless of how many references may become available. His name is already prominent in this relation, and deservedly so. . . . DR. GILLETTE'S "Pathological Types of Goitre" (page 137) contains the subject matter of his presentation under the same title at a meeting of the ASSOCIATION OF SURGEONS OF THE NEW YORK CENTRAL SYSTEM. . . . DR. VACCARO'S "Evaluation of Head Lesions" (page 140) is the first of two articles. The second, "Traumatic Injuries and Mental Disturbances," will appear in the May issue. . . . DR. SLOBE'S summary on "Injection Treatment of Hernia" (page 145) is another of those epitomes wherein the elements of value and authoritativeness seem inversely proportional to the number of words in which they are set forth. . . . DR. SEEGER, as chairman of the A. M. A. Council on Industrial Health, is in excellent position to be fully informed on "The Relationship of the Nurse in Industry to the Physician," concerning which (on page 146) he writes so interestingly. . . . DR. REUTER'S "Prevention of Industrial Dermatitis" (page 147), given at the Nurses' Symposium in Milwaukee, in February, summarizes a subject which is in the forefront of industrial medical attention

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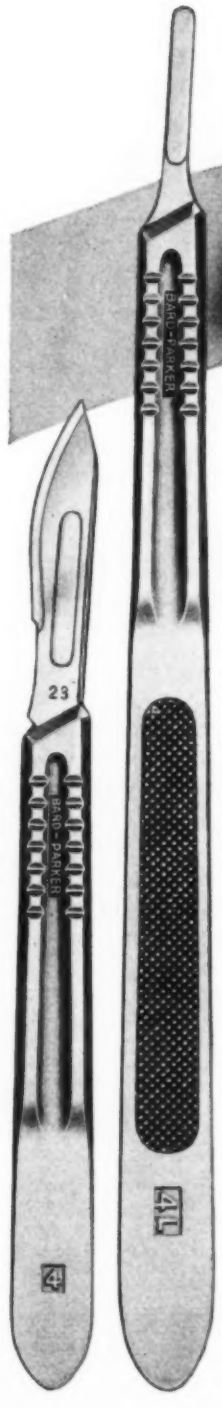

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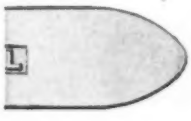

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at this time. .. . . AND MR. KUECHLE, in his analysis of "The Responsibility of the Industrial Nurse in the Efficient Administration of a Compensation Law" (page 150), read at the same symposium, speaks from profound experience with, and thorough knowledge in relation to, his subject. . . . MAX TRUMPER'S "Industrial Poisonings: Practical Aspects" (page 152) stresses, interestingly, the need to correlate clinical findings in an examination with a thorough occupational history. . . .

THEN comes the Complete Program of the Pittsburgh meeting, May 5 to 9, of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS and the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION (page 155). Where its mere entitlement is so eloquent of interest and quality, words of praise were supererogation. Suffice it to urge: DON'T MISS IT! . . . THE Proposed Amendments of Constitution and By-Laws of the A. A. I. P. & S., with their final appearance here (page 157)

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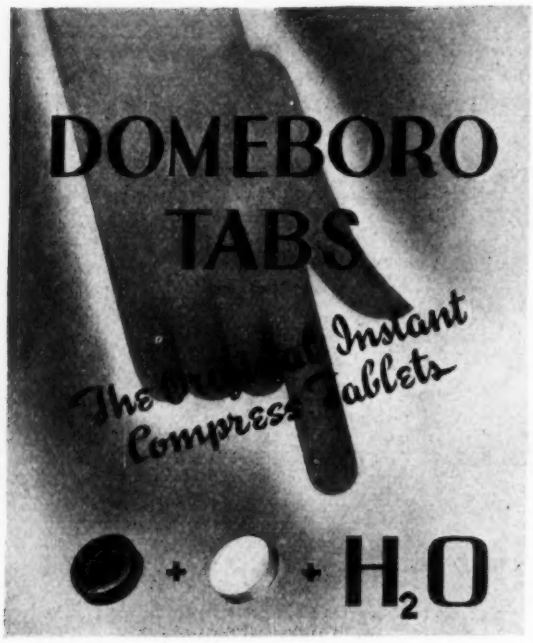

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—*Continued from Page 6.*

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DR. VONACHEN'S presentation of "The Case for a Health and Medical Plan" (page 157) not only presents, but also *proves!* In his usual forthright and sincere manner, he leaves no doubt that a "health program is a necessity for industries, large and small." . . .

THE SURGICAL ASSOCIATION of Atlanta and West Point Rail Road Company, the Western Railway of Alabama, and the Georgia Railroad, held its Twenty-First Annual Meeting at Atlanta, Georgia, March 20, 1941. "Importance of the Personality Aspect of Railroad Employees" was presented by DR. W. W. YOUNG, of Atlanta, guest speaker; "Significance of Acute Abdominal Pains," by DR. FLOYD W. MCRAE, of Atlanta; "Clinical Experience with Sulfathiazole," by DR. GORDON M. MYERS, of Detroit; and "Eye Symptoms Associated with General Diseases," by DR. HUGH M. LOKEY, of Atlanta. A Round Table was conducted by DR. J. R. GARNER, Chief Surgeon.

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ON THE News Front, first is the announcement from DR. LEROY U. GARDNER that a "Symposium on Tuberculosis in Industry" will be held at the Saranac Laboratory, June 9 to 14, 1941. The registration will be limited to 100, and the tuition is $25. Application should be addressed to Secretary, Trudeau School, P. O. Box 131, Saranac Lake, N. Y. The topics include: "The Disease, Tuberculosis — Incidence in Industrial Groups" — "Predisposing Factors, Theoretically Significant" — "Incidence and Causation of Tuberculosis, Various Industries" — and "Methods of Control."

THE following comes from PHILIP DRINKER: "Owing to the demand of the Government services and of the defense industries for physicians and engineers trained in industrial hygiene, the Harvard School of Public Health is planning to offer both its lecture and laboratory courses in this field, ordinarily given in the academic year, in a special summer course running from May 1 through July. Details may be secured from the Secretary of the School, 55 Shattuck Street, Boston, Massachusetts."

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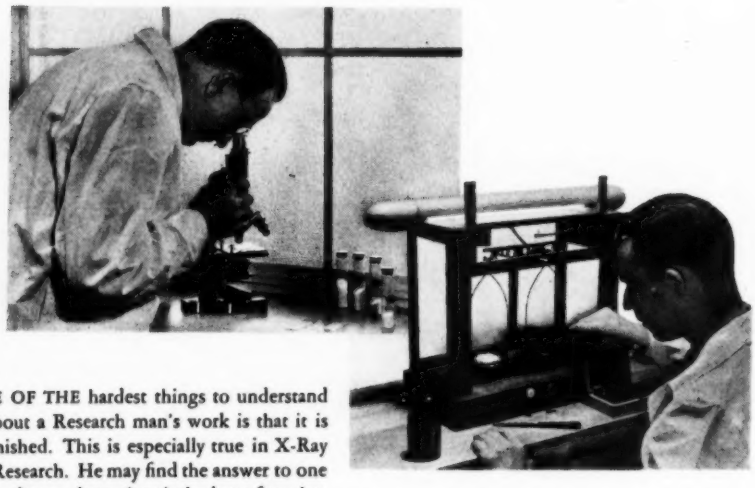

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trial doctor, under the heading "Opportunities in Industry," in a recent issue of *Medical Economics*, the author, HENRY P. MALMGREN, says: "Another medical director had these forthright remarks for the benefit of young doctors just starting industrial work: 'They must have the guts to take humiliation from laymen and medical busybodies; to be trampled underfoot by general practitioners who are always afraid the industrial man is going to treat a non-industrial case; to be able to laugh when they would like to kick hell out of things in general.' "

DR. HAYHURST accompanies his "Current Comment" on "Silicosis in Danish Industries" (page 159), with the following: " Space does not permit more than a word in reference to Professor de Bovis' translation, which includes no tables, but, as a groundwork for presenting the *Commentaries* below, the principal sub-titles concern: Part I., the x-ray examination and discussion of the stages of silicosis, its predisposing causes, symptoms and physical findings, differential diagnosis, evolution, prognosis, complications and treatment; Part II., a resumé of the personal researches of DR. CHRISTENSEN into 12 common industries, emphasizing comparative findings in the x-ray and clinical fields. In fact, DR. CHRISTENSEN'S researches of some 10 years ago are apparently the most comprehensive of attempts to compare the effects of inhaling siliceous dusts of different sources. It is to be noted, also, that in 1927, DR. CHARLES BADHAM distinguished differences in the radiographic appearances according to different dusts, for which he suggested the terms 'silicatosis' for silicates, and 'siliconosis' for the effects of mixed dusts of silica and silicates."

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NEW BOOKS

Surgery of the Hand

Review by

FREDERICK W. SLOBE, M.D., F.A.C.S.

SURGERY OF THE HAND, by JOHN HAROLD COUCH, M.A., M.B., F.R.C.S., Edin. Published by The University of Toronto Press, 1939.

THIS book is divided into two parts, the one devoted to injuries of the hand and the other to hand infections. The author quotes from DR. JOSEPH FULD of New York in the observation that "surgeons who do skillful work in other departments of surgery often fail to get equally good results in their work on hands." In discussing the correct first treatment the generally accepted principles of mechanical washing with soap and water, the avoidance of strong antiseptics, the use of debridement, and the removal of foreign material are emphasized.

The diagnosis of severed nerves and tendons is well illustrated by diagrams. A few tests are included which are not commonly known and are quite valuable. Following this the technique of repair of severed tendons and nerves is described, including a short section on the covering of raw surfaces and splinting.

In discussing tendon repair the four steps to a successful conclusion are: (1) recognition; (2) repair; (3) maintenance of repair; (4) motion. A division of any one of the 29 tendons and muscles in the hand causes impairment in the hand's efficiency unless the type of injury is diagnosed and correctly repaired. A valuable and simple rule is emphasized, "examine the hand, not the wound." It is the author's opinion that, if facilities for adequate

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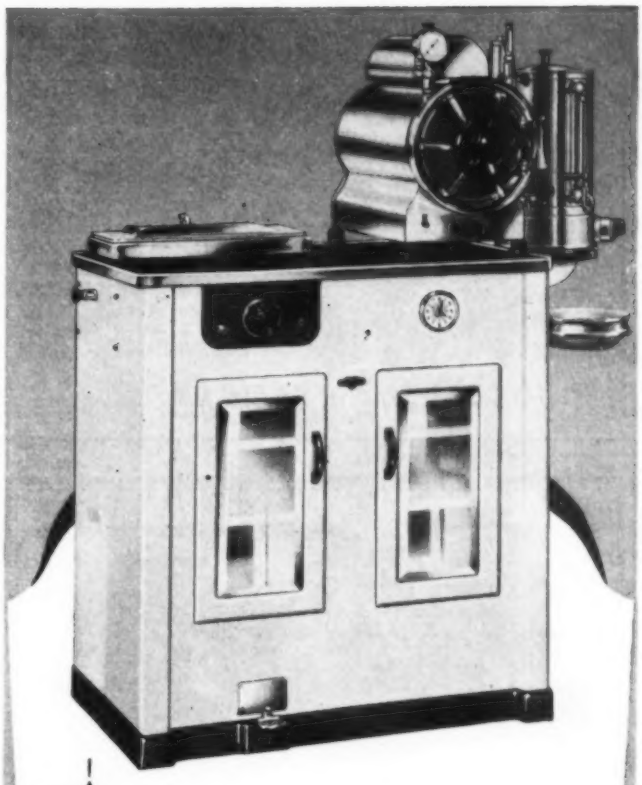

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repair are not available the skin should be closed in order to secure primary union, formal repair being carried out at a later date. This, no doubt, might be advantageous in isolated communities where too great a time interval would elapse before the patient could be brought to a suitable environment for primary repair. Obviously, if such an environment could be reached within a few hours the best procedure would be to leave the wound absolutely alone except for the application of a sterile dressing, bandage, and temporary splint.

After describing the technique of tendon suture, the importance of keeping the joints flexible during the healing period and the value of early active movement are emphasized.

Some very practical points are brought out in the discussion of amputation levels for the fingers and metacarpals. It is very unfortunate that compensation boards in some states make a hand case out of a finger case when the surgeon, instead of disarticulating the index or little finger at the metacarpo-phalangeal joint performs the far more functionally effective procedure of removing the distal one-third of the corresponding metacarpal. Much better service would be rendered to the injured employee were such defects in various compensation laws corrected. In finger amputation function rather than form should be the guiding factor. It is not sufficiently realized, for example, how useless any stump of the index finger is when the amputation level is proximal to the distal joint. Well emphasized, also, is the poor result usually obtained by disarticulation at the wrist.

The opening statement in the discussion of hand infections is a quotation from Dr. Lionel Auster, "I would let any house surgeon in New York take out my appendix at the end of his internship, but I don't think there are more than six men in New York whom I would trust with a hand infection." An infected finger often causes a disability period five times as long as that following a cholecystectomy.

The author describes the treatment of paronychia, felon, inflammation in the tendon sheaths, and cellulitis with

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descriptions of the operative procedures when indicated and with illustrations describing incisions made. The methods employed by the author, in general, are most commendable. Strangely, no mention is made of infections of the lumbrical space, mid-palmar space, thenar space, etc.

The author quotes from the Workmen's Compensation Board of Ontario in giving an industrial definition of infection as follows: "Infection is the state of affairs which exists when the seriousness of the case becomes due to the immediate or subsequent introduction of bacteria rather than to the nature of the original injury." This Board has kept a registry of injured hands for the past 20 years. A group of 1500 cases of infected hands was subdivided into three divisions: 1470 were temporarily disabled from work but ultimately returned to their original jobs; 25 were totally and permanently disabled; five proved fatal. Of those temporarily disabled 44,000 working days were lost with a compensation outlay of about $113,000. The 25 totally and permanently disabled had a total period of compensation of 3,625 days lost with $75,750 paid out. Grouping all three divisions together, hand infections in Ontario cost about 48,000 working days yearly and nearly a quarter of a million dollars without emphasizing the permanent disabilities and deaths. Much of this is preventable since the occurrence of infection usually suggests negligence either concerning the prevention of the original injury or failure to obtain prompt or correct treatment.

The author has condensed considerable material in the 147 pages in this book. There are many full-page illustrations which, though diagrammatic, are extremely clear and instructive. The book is intensely practical and describes those principles and procedures which in the author's hands have yielded the best results. Presenting a concise, clear description of the diagnosis and treatment of the more common injuries and infections of the hand it should hold definite appeal for those interested in either general or traumatic surgery.

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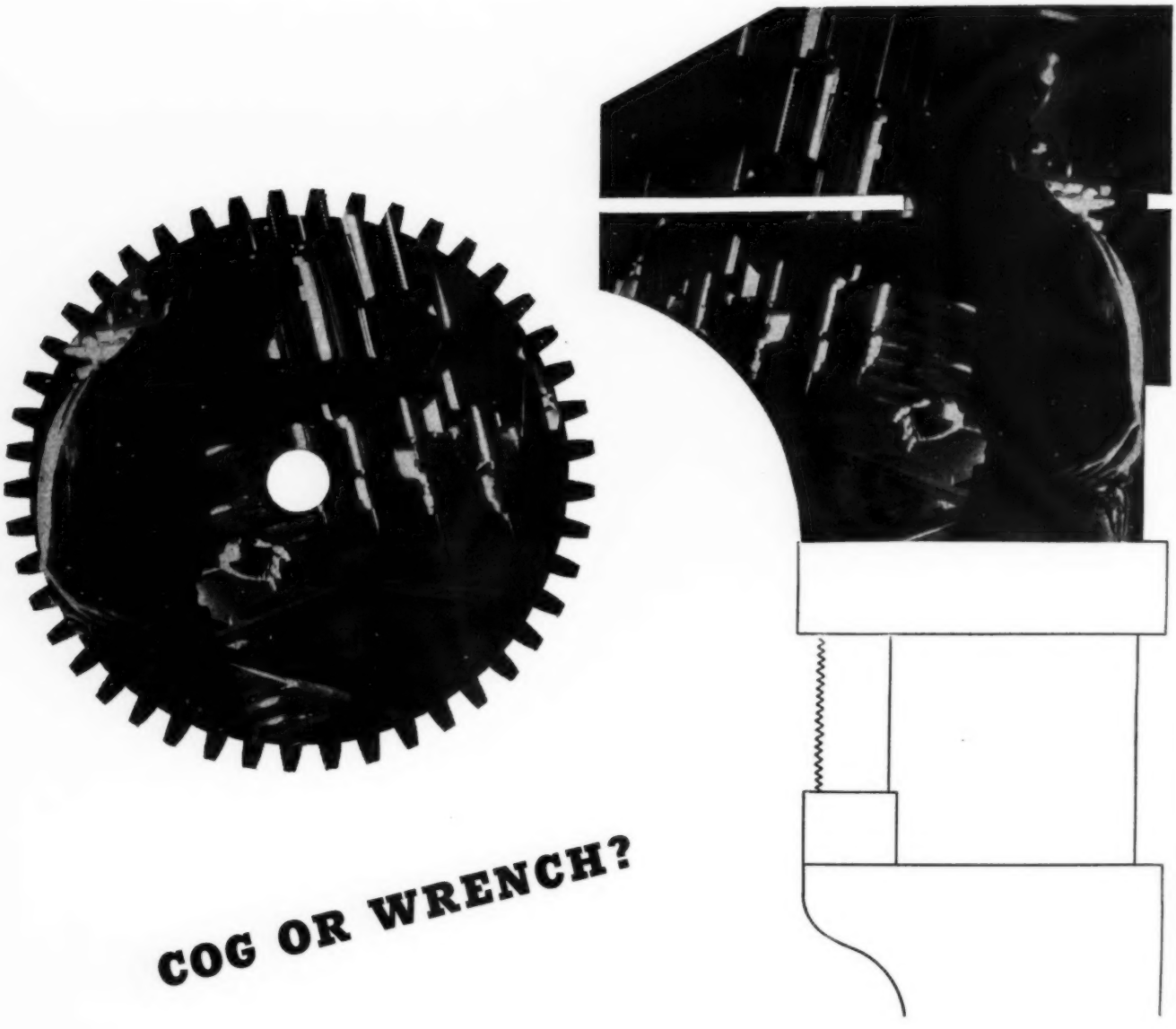

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The Science, the Law and the Economics of Industrial Health

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Sulfathiazole

—*Present Status in the Treatment of Infections*—

CHARLES H. RAMMELKAMP, M.D.,
Boston, Massachusetts

SEVERAL years have passed since the introduction of sulfanilamide, and although it is now well recognized that this drug is an effective chemotherapeutic agent in certain diseases, in other infections, uncertainty as to its beneficial action still exists. It is established that sulfanilamide has lowered the mortality in streptococcal and meningococcal diseases. It has been very effective in the treatment of gonococcal infections and in certain of the infections of the urinary tract. There are some infections, however, that respond poorly, if at all, to the administration of this drug. In addition, sulfanilamide produces several undesirable side reactions such as nausea, vomiting, cyanosis, fever, rashes, agranulocytosis and hemolytic anemia. It is not surprising, then, that numerous derivatives of sulfanilamide have been prepared and tested for their chemotherapeutic and toxic properties. Sulfapyridine, one of these derivatives, has been found to be much superior to sulfanilamide in the treatment of pneumococcic infections. It is also effective in the treatment of infections due to the gonococcus. Sulfapyridine, in turn, has failed to alter significantly the course of some infections. In general, the toxic manifestations of sulfapyridine are similar to those caused by sulfanilamide except that nausea and vomiting, and hematuria occur much more frequently.

In April, 1939, Fosbinder and Walter[1] described several thiazole derivatives of sulfanilamide. Two of these derivatives, sulfathiazole and sulfamethylthiazole, were found to possess bacteriostatic properties *in vitro*. Since that time considerable laboratory and clinical evidence has accumulated concerning the beneficial action of these drugs, and recently sulfathiazole has been released to the medical profession. It is well, therefore, to examine the results of these studies before using this drug clinically.

Absorption, Excretion and Diffusion

BEFORE discussing the use of sulfathiazole in the treatment of various infections, it might be well to review, briefly, the results of studies on the absorption, excretion, diffusion and toxicity of this drug. Studies in the experimental animal[2,3] and in man[2,4,5] have demonstrated that sulfathiazole is more readily absorbed and more rapidly excreted than sulfapyridine. When four grams of the drug are given as a single oral dose to normal individuals, the concentration of sulfathiazole in the blood is found to reach its maximum level about two to four hours later. During the next few hours there is a rapid decrease in concentration and at the end of 24 hours little or no drug remains in the blood. If the excretion of the drug in the urine is followed at the same time, about 40% of the ingested sulfathiazole will be found in the urine after six hours, and from 70 to 90% will be found in the 24-hour urine. When sulfathiazole is administered continuously in doses of one gram every four hours, an average concentration of about 5 mg. per 100 cc. is maintained. Under a similar schedule the level of the drug in the urine varies from 35 to 450 mg. per 100 cc. It is important to remember, however, that individuals vary considerably in their ability to absorb and to excrete sulfathiazole. Vomiting and diarrhea decrease absorption whereas cardiac failure and poor renal function cause decreased excretion. In such patients the concentration in the blood must be followed closely. If necessary, sulfathiazole may be administered as the sodium salt intravenously. It is perhaps important to emphasize that absorption is poor when sulfathiazole is given by rectum.

From the Evans Memorial, Massachusetts Memorial Hospitals, and the Department of Medicine, Boston University School of Medicine, Boston, Massachusetts; address given before the NEW YORK STATE SOCIETY OF INDUSTRIAL MEDICINE, Utica, New York, October 31, 1940.

Sulfathiazole is less readily acetylated than sulfapyridine, indeed, in this respect it is comparable to sulfanilamide. The average concentration of acetylated sulfathiazole present in the blood has been found to be about 12%, whereas 50% of the total concentration of sulfapyridine is usually acetylated.[6] The amount of conjugated sulfathiazole present in the urine is usually about 25% which is considerably less than the amount found after sulfapyridine administration. It must be remembered, however, that acetylsulfathiazole is very insoluble so that examination of the urine of patients receiving sulfathiazole will frequently show crystals.

It has been reported[5] that sulfathiazole diffuses readily into pleural and ascitic fluids, although a definite lag in the inward and outward diffusion has been noted. It is interesting, however, that little sulfathiaozle diffuses into the spinal fluid, although occasionally adequate levels are observed. It is because of this irregular diffusion of the drug into the spinal fluid that meningitis cases have not been treated with sulfathiazole.

Toxic Manifestations

ALTHOUGH sulfathiazole has been found to be about one-third more toxic for mice than sulfanilamide[1], it is only half as toxic for the same animal as sulfapyridine and sulfamethylthiazole. It has been noted, also, that hematuria with accumulation of acetyl-sulfathiazole crystals in the kidneys and ureters occurs in the experimental animal. In man the toxic symptoms and signs have been studied,[7,8] some of which are important to recognize early. One of the most frequent toxic symptoms observed following sulfapyridine therapy is nausea and vomiting, indeed, about 80% of patients complain of nausea. It can be seen from Table I that the incidence of nausea is much less following sulfathiazole administration. Vomiting, when it occurs, is usually not severe. Hematuria occurs less frequently in the patients treated with sulfathiazole; nevertheless, it is important to examine the urine for blood, since this is the first sign of renal irritation due to the accumulation of crystals. When hematuria occurs, the drug should be discontinued and fluids forced.

TABLE 1.
TOXIC MANIFESTATIONS OF SULFATHIAZOLE

	Per cent
Nausea	35
Vomiting	29
Hematuria	5
Skin eruptions	5
Drug fever	3
Anemia	3
Psychosis	1

Rashes and fever occur more frequently than in sulfapyridine treated cases. Usually the rashes are accompanied by fever, although either may occur separately. The figures quoted in Table I are probably too low in that the majority of these cases received the drug for less than one week, whereas rashes and fever commonly occur between the eighth and twelfth days of therapy. Haviland and Long[9] observed skin eruptions in 12% of their cases. The most common skin eruption is an erythema-nodosum-like lesion, occurring especially over the anterior surface of the legs, around the elbows and under the eyes. These lesions may be extremely sensitive. There is usually an associated injection of the conjunctivae and frequently injection of the sclerae. We have observed one case where there was actual hemorrhage into the sclerae. Frequently an erysipeloid-like lesion appears over the nose and cheeks; this also may be extremely sensitive. In addition, generalized maculo-papular eruptions and urticarial-like lesions have been observed. The rash and fever disappear rapidly following the withdrawal of the drug, and occasionally disappear even though the drug is continued. In one of our cases, the rash disappeared under therapy only to recur about a month later.

Another important toxic sign is the development of anemia. We have seen no case of acute hemolytic anemia, however, about 3% of the cases will show a drop of from 20 to 40% in the hemoglobin concentration. No cases of agranulocytosis have been reported, although leucopenia has been observed occasionally.

Pneumococcic Infections

EXPERIMENTAL studies[10,11,12,13] have demonstrated that sulfathiazole and sulfapyridine are about equally effective against pneumococci both *in vitro* and in the experimental animal. Reports on the use of sulfthiazole in the treatment of pneumococcal infections in man are limited to cases of lobar pneumonia. Fig. 1 illustrates the typical response observed following treatment with sulfathiazole. This case, a girl, 26 years of age, entered the hospital complaining of pain in the left chest of 12 hours' duration. She had few signs of consolidation, however, x-ray showed a definite infiltration of the left lower lobe. The sputum and blood both yielded type XII pneumococci on culture. Sulfathiazole was administer-

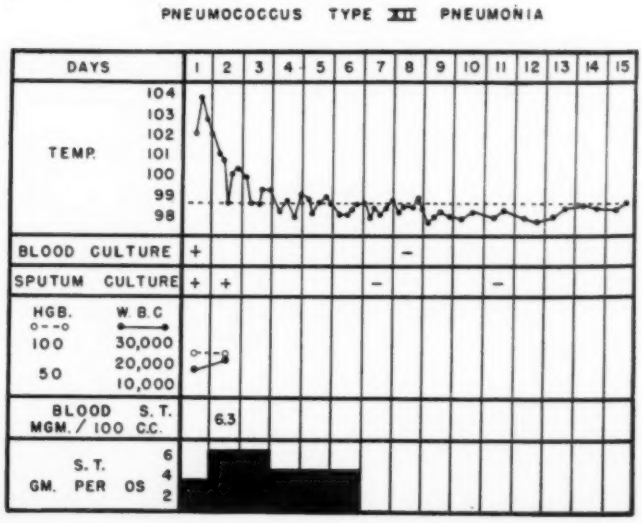

Fig. 1.
S.T. = Sulfathiazole

ed, following which there was a rapid fall in temperature and clinical recovery.

Recently Finland, Lowell and Strauss[8] reported a mortality of 11.5% in a series of 104 cases of lobar pneumonia treated with sulfathiazole. During the same season they treated 382 cases of pneumonia with sulfapyridine with a mortality of 12.6%. They observed that the clinical response in the two series of cases was almost identical and conclude that sulfathiazole and sulfapyridine are equally effective in the treatment of lobar pneumonia. Flippin, Schwartz and Rose[7] treated 100 cases of pneumonia with sulfathiazole and 100 cases with sulfapyridine. Their uncorrected mortality rate for the sulfathiazole treated cases was 12%, for the sulfapyridine treated cases was 15%. In their series they felt that sulfapyridine brought the temperature down to normal more rapidly than sulfathiazole, however, the severity of the nausea and vomiting in the patients receiving sulfathiazole was less than those receiving sulfapyridine.

Pneumococci have been found to vary somewhat in their susceptibility to the sulfonamide drugs, so that recently the term *drug fastness* has been coined to describe these strains. It has been found that pneumococcus types II, III and V are likely to have more resistant strains than other types. In addition, *drug fastness* has been observed to develop during chemotherapy[8] which may account for the poor response seen in some cases. Because we now have a new drug which is effective against the pneumococcus, it is important to emphasize that Finland, Lowell and Strauss[8] have found that strains of pneumococci which are resistant to sulfapyridine are about equally resistant to sulfathiazole and sulfamethylthiazole. Cases which do not respond to either sulfathiazole or sulfapyridine should be given type-specific serum.

Staphylococcal Infections

SOON after the thiazole derivatives were described two cases of *Staphylococcus aureus* bacteremia which recovered following treatment with sulfamethylthiazole were reported.[14,15] At about the same time the thiazole compounds were found to inhibit the growth of staphylococci in broth[12,13] and in urine[16,17] cultures. Because of these early favorable results the lay press gave these compounds considerable publicity, and during the past few months further instances of recovery following therapy with sulfamethylthiazole and sulfathiazole have been reported.[18,19,20,21] It should be pointed out that similar results were reported following the use of sulfanilamide and sulfapyridine in these infections.[22,23,24] It is now believed, however, that sulfanilamide and sulfapyridine have little effect on the course of *Staphylococcus aureus* bacteremia.[25]

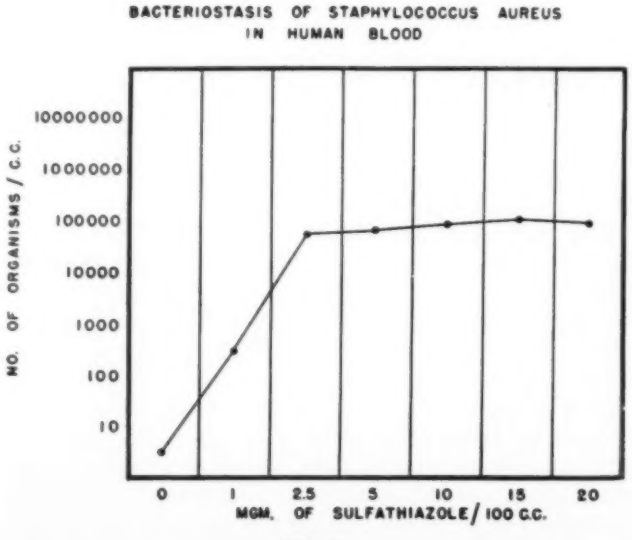

Fig. 2.
Representing the results obtained in 10 experiments on 10 samples of blood

Before using the thiazole compounds in the treatment of staphylococcal infections, we studied the action of the various sulfonamide drugs in human defibrinated blood. The technique of these experiments is reported in detail elsewhere.[26] In brief, the various drugs are added to defibrinated whole blood following which pathogenic staphylococci are added in varying numbers. The inoculated tubes are sealed and placed in a rotator at 37°C for 24 to 48 hours. The tubes are then removed and those cultures showing no hemolysis are plated out and the colonies counted.

We were first interested in determining the concentration of sulfathiazole necessary to obtain the maximal effect. Fig. 2 shows the effect of various concentrations of sulfathiazole on the degree of *bacteriostasis* in human blood. The solid dots represent the largest inoculum of *Staphylococcus aureus* which failed to cause hemolysis after 48 hours' incubation. It can be seen from this chart that a concentration between 2.5 and 5 mgm. per cent is necessary to obtain maximal bacteriostasis. Increasing the concentration of sulfathiazole above this level does not increase the *bacteriostatic* action appreciably.

TABLE 2.
COMPARATIVE ACTION OF VARIOUS DRUGS ON STAPHYLOCOCCUS AUREUS IN HUMAN BLOOD

	Inoculum Bacteria/cc	48 hours' incubation Bacteria/cc
Control	3,200	100,000,000
Sulfanilamide*	3,200	100,000,000
Sulfapyridine	3,200	804
Sulfamethylthiazole	3,200	20
Sulfathiazole	3,200	0

* Concentration of drugs is 10 mgm. per cent.

When the *bactericidal* action of sulfathiazole in blood is studied, similar results are obtained. Concentrations of sulfathiazole of 1 mgm. per cent cause little or no killing, whereas blood containing between 2.5 and 5 mgm. per cent will kill over 1,000 staphylococci per cubic centimeter.

In Table II the action of the various drugs in concentrations of 10 mgm. per cent is compared. This represents the typical result obtained from numerous experiments. It is demonstrated quite dramatically that sulfanilamide has little or no

effect on the growth of the staphylococcus. Sulfapyridine caused some bacteriostasis when the inoculum contained less than 10,000 organisms per cubic centimeter of blood. Sulfathiazole and sulfamethylthiazole containing cultures exhibited not only a bacteriostatic action but also a bactericidal effect. Sulfathiazole was somewhat more effective than sulfamethylthiazole. Sulfathiazole caused some killing when the blood contained over 100,000 organisms per cubic centimeter. This increase in the bactericidal effect of the blood can be observed when sulfathiazole is given by mouth to normal individuals.

Recently we made a study of seven patients with *Staphylococcus aureus* bacteremia treated with sulfathiazole.[27] Three of these patients recovered. No conclusions can be drawn from such a small series of cases, but certain problems deserve special comment.

One of the patients who failed to recover was a 17 year old boy who had osteomyelitis of the sacro-iliac joint. On admission to the hospital his temperature was 102°. Physical examination revealed local tenderness over the left sacro-iliac joint. The leucocyte count was elevated to 20,000 and cultures of the blood showed *Staphylococcus aureus*. Roentgenograms of the pelvis showed no abnormality.

This patient first received sulfapyridine which failed to lower the temperature or to sterilize the blood stream. Sulfathiazole was then administered following which the blood became sterile. The temperature remained elevated and x-rays taken at this time now showed definite involvement of the sacro-iliac joint. The local lesion was drained and the temperature immediately returned to normal. During the third hospital week the temperature again became elevated; however, cultures of the blood remained sterile. Sulfathiazole therapy had no apparent beneficial effect at this time. During the eleventh hospital week the sacro-iliac region was again explored and a large collection of pus was found below the area originally drained. During the exploration considerable bleeding was encountered. Attempts to control the bleeding after operation were of no avail and the patient expired a week later following a massive hemorrhage.

The clinical course of this case serves to emphasize several important points. Originally he received sulfapyridine which failed to sterilize the blood. When sulfathiazole was administered, the blood immediately became sterile. Studies made at the time of sulfathiazole administration showed that there was an increase in the bactericidal power of the blood. In spite of chemotherapy, the temperature remained elevated, and it was not until the local lesion had been drained that the patient became afebrile. Later, he again developed fever without an associated bacteremia. Continued elevation in temperature in patients with staphylococcal infections usually indicates further abscess formation. In this case a second abscess was found at operation, a severe hemorrhage was encountered and the patient expired.

A second case was that of a 40-year-old man who entered the hospital because of pain in the chest. A month prior to entry he fractured the right femur and was treated by a Roger-Anderson reduction. The pins which had been placed through the femur were removed two weeks later. Several days before entry he developed bilateral chest pain and experienced several chills. He also noticed some pus around the upper part of the cast.

At the time of entry to the hospital he was found to have rales and dullness to percussion at the base of the left lung. Considerable pus was noted around the upper pin hole in the right thigh. X-rays of the chest revealed some consolidation of the lung at the left base. The leucocyte count was elevated and cultures of the blood showed *Staphylococcus aureus*. Sulfathiazole therapy was instituted at the time of admission following which all blood cultures were sterile. Thoracentesis on the third and fourth hospital days yielded a turbid fluid which showed staphylococci on culture. Later chest taps were sterile and surgical drainage was not necessary. In spite of chemotherapy the fever continued and it was not until adequate drainage of the right femur was established that the patient recovered.

This case was especially interesting in that the staphylococcal empyema became sterile during sulfathiazole therapy. It is unusual for chemotherapy to sterilize local lesions. In general, it may be stated that surgical drainage of the local abscess is necessary for recovery in most cases.

In summary, then, these *in vitro* and clinical studies show that sulfathiazole is superior to sulfanilamide, sulfapyridine and sulfamethylthiazole in its effect against *Staphylococcus aureus*. When sulfathiazole is given to patients with staphylococcal bacteremia, the number of organisms in the circulating blood is decreased. This action is valuable in the prevention of metastatic abscess formation during the period of blood stream invasion. It has been pointed out[28] that 82% of all cases of staphylococcal bacteremia develop secondary lesions, and it is the development of these metastatic abscesses which can not be drained that determines the fatal outcome in the majority of instances.

Infections of the Urinary Tract

SULFANILAMIDE has been used extensively in the treatment of infections of the urinary tract, and has proved to be effective in the majority of instances.[29,30] *B. coli*, the organism most frequently associated with this infection, has been found to be susceptible to high concentrations of sulfanilamide. Other organisms which are not uncommonly the cause of these infections are *B. proteus, B. lactis aerogenes, Streptococcus fecalis,* and *Staphylococcus aureus*. Infections due to these organisms respond poorly to therapy with sulfanilamide. Further, Helmholz[31] reports that *Streptococcus fecalis* is not affected by either sulfanilamide or sulfapyridine.

In preliminary experiments sulfathiazole and

sulfamethylthiazole were found to kill small inocula of *Streptococcus fecalis, Staphylococcus aureus, B. proteus* and *B. coli* in urine.[31] The growth of these organisms can also be inhibited in broth cultures.[13] These results suggested that sulfathiazole might be useful in the treatment of infections of the urinary tract.

Recently we made a study of 25 patients with infections of the urinary tract treated with sulfathiazole.[16] Briefly, the study was conducted as follows. All samples of urine were catheterized in the females, clean-voided in the males. Specimens were obtained before treatment, two days after the institution of therapy, and at varying intervals thereafter. Cultures, a determination of the concentration of sulfathiazole, and a study of the urinary sediment were made on each specimen. The majority of patients received two grams of sulfathiazole by mouth, followed by one gram every four hours. The total duration of treatment varied from four to 20 days. The level of free sulfathiazole obtained in the urine varied from 32 to 456 mgm. per 100 cc.

In order to evaluate further the efficacy of sulfathiazole therapy in these infections we made certain *in vitro* observations. These *in vitro* studies were carried out using as media the urine obtained before therapy and after two days of treatment. A 12-hour culture of the organism isolated previously was added to the two samples of urine and the degree of bacteriostasis followed during 24 hours' incubation. In addition, a comparative study of the action of the sulfonamide drugs in concentrations of 10 mgm, per 100 cc. was made on the organisms isolated from these patients.

B. Coli Infections

THERE were 20 cases of *B. coli* infections. Thirteen of these cases were classified as cured, in that the urine cultures became sterile, the temperature returned to normal, the urine became clear and all symptoms were relieved. The following case illustrates the typical response observed in the patients classified as cured.

A 56-year-old woman entered with a history of dysuria of three weeks' duration. On admission she had a chill, and examination at that time revealed tenderness over both costo-vertebral angles. The urine and blood cultures contained *B. coli*. The urinary sediment contained many leucocytes. The response to sulfathiazole therapy was indeed dramatic, some subjective improvement being noted in the first 24 hours. The temperature returned to normal, cultures of the blood and the urine became sterile and the number of cellular elements in the urine decreased.

In vitro studies of this patient's urine before and during therapy showed that the urine containing sulfathiazole was bactericidal. It was found that over 10,000 *B. coli* per cubic centimeter of urine were completely killed within 24 hours, whereas the organisms grew freely in the urine used as the control. This is the result observed in the majority of patients with *B. coli* infections when the urine is studied before and during therapy with sulfathiazole.

Comparative studies using sulfanilamide, sulfapyridine, sulfamethylthiazole and sulfathiazole in concentrations of 10 mgm. per 100 cc. against the various strains of *B. coli* isolated from these patients showed that sulfathiazole was somewhat superior. Similar results were obtained when *B. lactis aerogenes* was used as the test organism.

Of the remaining seven cases of *B. coli* infections, five were classified as improved and two as failures. The five cases which improved under therapy were all complicated; two were diabetics, one had prostatic obstruction with an inlying catheter, one had a large non-functioning kidney, and another was insufficiently treated. All of these cases improved under therapy, but were not cured. The two cases classified as failures included one case of impacted ureteral calculus and the other case was improving, when, following a genito-urinary procedure, he suddenly went into shock and died.

B. Proteus Infections

THERE were three cases of *B. proteus* infections, two of which responded immediately. The third case had a supra-pubic cystostomy wound and multiple lung abscesses. Under therapy the urine became sterile, only to become infected again when he was removed from the hospital.

The first case was admitted with an atonic bladder, flaccid paralysis of the left arm and leg and a left Horner's syndrome. A definite diagnosis was not made, but the signs were thought to be due to a vascular lesion of the spinal cord. She was placed on tidal drainage until her forty-fourth hospital day when she regained function of her bladder. At times she ran a low-grade fever and complained of burning on urination. The urine contained many leucocytes and yielded *B. proteus* on culture. She was given two courses of sulfanilamide without apparent benefit. Two days after the institution of sulfathiazole therapy the urine became sterile and has remained sterile. Another patient, 70 years of age, had a vesico-vaginal fistula with symptoms of cystitis for many months. The usual forms of therapy failed to relieve her symptoms. She was given 0.5 gm. of sulfathiazole three times daily for one week. Her urine became sterile and all symptoms were relieved.

In vitro studies using the organisms isolated from these cases demonstrated that sulfanilamide had little effect on the growth of *B. proteus*, whereas sulfathiazole caused marked bacteriostasis.

Staphylococcus Aureus Infections

THERE were two cases of staphylococcus aureus infections. In one case the urine became sterile, but the fever continued. Following drainage of a perinephritic abscess the patient recovered. In the second case the urine was sterile on several occasions during therapy, and the number of leucocytes decreased in the urine. This case was complicated by pyonephrosis, megaloureters, and

multiple lung abscesses, and classified as a failure.

Again, *in vitro* studies using urine as the media showed that sulfathiazole and sulfamethylthiazole caused marked inhibition or killing, whereas sulfanilamide failed to affect the growth of staphylococci.

In summary, then, sulfathiazole has been found to be effective in the treatment of infections of the urinary tract due to *B. coli, B. proteus* and *Staphylococcus aureus.* Some complicating factor was present in those cases which did not respond immediately. *In vitro* studies have demonstrated that sulfathiazole is more effective than sulfanilamide against *B. coli, B. lactis aerogenes, B. proteus, Staphylococcus aureus* and *Streptococcus fecalis.* These results warrant further clinical trial of sulfathiazole in treating urinary infections.

Miscellaneous Infections

THE value of sulfathiazole in the treatment of other infections is not known at the present time. Sulfathiazole is reported to be as effective as sulfanilamide and sulfapyridine against hemolytic streptococci both *in vitro* and in the experimental animal.[12,13,32,33] In one report, sulfathiazole in concentrations of 1 mgm. per 100 cc. was found to be more effective against gonococci than other sulfonamide compounds.[12] Clinical reports concerning the use of sulfathiazole for the treatment of gonococcal infections are encouraging.[34] *In vitro* experiments have shown that sulfathiazole is slightly superior to sulfapyridine and sulfanilamide against *S. suipestifer* and organisms of the typhoid-dysentery group.[35,36]

Conclusions

SULFATHIAZOLE is rapidly absorbed from the gastro-intestinal tract and is rapidly excreted by the kidneys. Relatively small amounts of the drug are acetylated. Diffusion into pleural and ascitic fluids takes place readily, whereas diffusion into the spinal fluid occurs irregularly.

Sulfathiazole causes less nausea, vomiting and hematuria than sulfapyridine; however, skin rashes occur more frequently.

Sulfathiazole has been used in the treatment of lobar pneumonia. The results compare favorably with sulfapyridine treated cases.

Sulfathiazole therapy of *Staphylococcus aureus* bacteremia has given encouraging results. Administration of this drug causes an increase in the bactericidal action of the blood and decreases the number of circulating organisms. This action is most desirable in the prevention of metastatic abscesses during the period of blood stream invasion. Surgical drainage of the localized abscesses is essential for recovery.

The use of sulfathiazole in the treatment of infections of the urinary tract has been found valuable. It is the only sulfonamide compound which affects *Streptococcus fecalis,* and, in addition, it is probably somewhat more effective than sulfanilamide against infections due to *Staphylococcus aureus, B. porteus, B. coli* and *B. lactis aerogenes.*

A concentration between 3 and 5 mgm. per 100 cc. of sulfathiazole should be maintained in the blood in staphylococcal and pneumococcal infections. In the average adult case an initial dose of two grams followed by one gram every four hours is sufficient to maintain such concentrations. In the treatment of urinary infections smaller amounts of sulfathiazole may be used.

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Pathological Types of Goitre

—*The Characteristics and Results of Treatment*—

Norris W. Gillette, A.B., M.D., F.A.C.S.,
Toledo, Ohio

THE American Association for the Study of Goitre has classified goitre as follows:

1. Smooth non-toxic goitre.
2. Smooth toxic goitre.
3. Nodular non-toxic goitre.
4. Nodular toxic goitre.

This simple but comprehensive classification was promulgated in order to bring some unanimity of opinion in the classification of goitre among the various groups interested in the subject. In no sense does it describe the pathological histology in the microscopical sections, but it does provide microscopical pictures which usually give an idea of the clinical entity.

The smooth, non-toxic goitre suggests the endemic or colloidal type which is so commonly present in the adolescent.

The smooth, toxic goitre suggests either the endemic type which has become toxic, or the typical exophthalmic goitre or Grave's disease which is usually symmetrically enlarged unless complicated with adenoma or cysts.

The nodular non-toxic type suggests the non-toxic adenoma which is a simple adenoma superimposed upon colloidal goitre before it has become toxic, which so frequently occurs, or the cystic or calcified goiter, while the nodular toxic goitre suggests the adenoma or the cystic goitre which has progressed far enough for toxic symptoms.

If we wish, however, to exactly identify a goitre, we must look at the histological picture of it and by its microscopical appearance we can in most cases give a reasonably accurate estimation of what damage has been done to the patient and what the future would have been if the goitre had been left in situ. While different nomenclatures have been used by clinics, the best in my opinion is as follows:

1. Simple goitre or endemic goitre indicating an early enlargement of the thyroid characterized by an increase in the size of the acini which are dilated with colloidal material. The cells lining the acini are somewhat flattened due to the pressure of the colloid, but otherwise are unchanged.

2. Adenomatous goiter is characterized by definite adenomas superimposed upon a colloidal background and evolving out of it by hypertrophy of the acinal cells. This type of goitre may either be non-toxic or toxic.

3. Grave's disease or exophthalmic goitre presents a picture, when the patient has not been treated with iodine, of papillary projections into the acini which are definite and unmistakable, but when iodine or its derivatives have been administered the papillae will quickly smooth down so that the picture is one of colloidal goitre and the diagnosis may be made by a simple small papilla. Since iodine is so frequently taken by goitre patients we seldom see the textbook picture of Grave's disease anymore.

4. Grave's constitution is a pathological picture of a type of goitre identified by the late Dr. A. S. Warthin. It consists of lymph nodules irregularly disseminated through the thyroid structure with lymph cells spreading from the nodes. In addition to this there is a generalized increase in lymph structure throughout the body. The colloid has to a great extent been replaced by cellular structure.

5. Focal adenoma appears as broken up and irregular cellular tissues which are piled up into nodular areas and simulate microscopically colloidal adenomas. This type of goitre is very prone to develop into malignancy.

6. Riedel's struma, struma lymphomatosis or Hashimoto's disease which are to all intents and purposes the same, present the picture of a structure markedly fibrous, containing few thyroid cells and evidencing clinically no increase in toxicity and may even show a hypothyroidism. Owing to the fibrous structure which may contract and constrict, there may develop a marked stridor due to the pressure on the trachea.

7. Malignancy usually develops from adenomas and the percentage is great enough to make it a real danger. Microscopically it can not be told from adenoma until it begins to spread outside of the thyroid capsule when it is recognized as such, or until it has already metastasized. Various types of malignancy are recognized, the commonest types being the adeno-carcinoma and the papillary carcinoma. The fact that carcinoma may be developing in any adenomatous goitre and is present in approximately one in 20 cases makes it a real danger to disregard the nodular goitre even if the toxicity is low. In endemic areas which are those containing little or no iodine in the soil, the normal thyroid begins to enlarge during the adolescent period, the enlargement being due to an increase in the colloid in the acini. In spite of our immense amount of research no adequate reason for this is known. It is well known, however, that small amounts of iodine taken in early childhood will to a large extent prevent the development of endemic goitre. It has been suggested that goitre is an infectious disease and the iodine acts by killing the organism of a parasite which is the etiological factor. Opposed to that is the view that iodine is necessary to the normal evolution of thyroid structure and production of the thyroid secretion or thyroxin which contains iodine. In any case the goitre started by the lack of iodine in the system. If iodine is administered in any form during the period of simple colloidal enlargement a definite reduction in the size of the goitre will usually ensue and if the treatment is continued carefully over a long period the cure may be permanent. As I stated before the adenomas develop out of the colloidal enlargement of the thyroid and in the beginning they are small and either non-toxic or relatively so. They may

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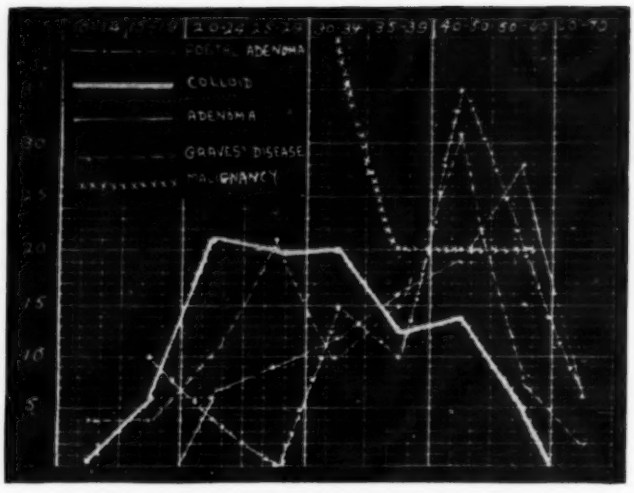
continue quiescent over a long period of time until some infection, stress, strain, or physiological function activates them, or they may be toxic from the beginning. The administration of iodine to this type of goitre is fraught with some danger. The patient will appear to do well for a period and then we are suddenly awakened to the fact that all of the symptoms have returned with increased virulence. In the non-toxic type of adenoma which has lasted for years the iodine must be administered with great care.

The most vivid example that I have seen of what iodine may do to a quiescent goitre was that of a man 70 years of age who had a very large nodular goitre which had only troubled him from a cosmetic standpoint. This goitre had been present for 30 years. He was a preacher in a small town and one of his parishoners advised him to take a patent drug advertised to cure goitre.

This medicine contained a large amount of iodine. After taking it for two weeks he began to lose weight and within 40 days had lost 30 pounds. His heart began to palpitate and he developed all the signs and symptoms of a toxic goitre.

The use of iodinized salt has undoubtedly cut down the incidence of goitre and it has also probably activated some non-toxic goitres. The appearance of a large number of small but very active goitres may be accounted for in this manner. The good, however, will far outweigh the evil. Kimball analyzed all of the brands of iodinized salt on the market to determine their iodine content and found that there was a wide variation in them from their advertised content and that two much used brands contained only a trace of iodine. Due to overlapping it is, of course, impossible to tell the results of this deficiency in any community.

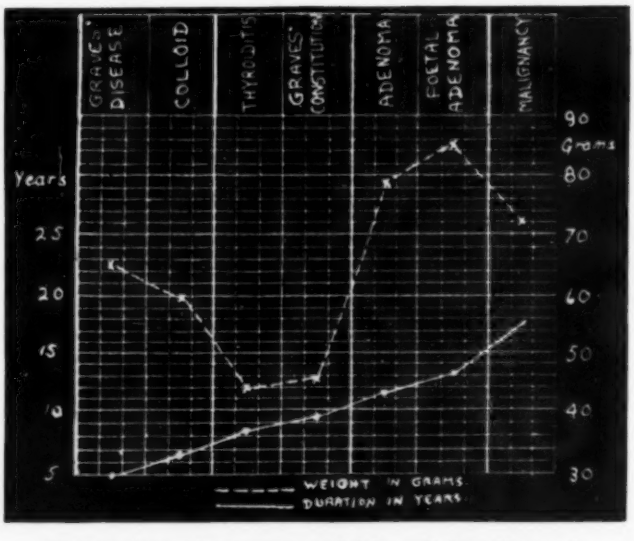
The only excuse for operation of an adolescent or endemic goitre is pressure on the trachea or exceptionally for cosmetic reasons. The treatment of the endemic goitre patient with some form of iodine carefully regulated according to the reaction of the patient should be continued over a period of months or years if it remains non-toxic, or only mildly so. In the case of adenomatous goitre, however, the thyroid should be removed, first, because nearly all of them become toxic in time and early removal prevents damage to the heart, nervous system, and other body structures, and secondly because of the above mentioned danger of malignancy. It must be noted that if the toxicity continues over a long enough period the damage that is done will be permanent and removal of the goitre will not repair the damage done. It only stops further damage. The operative mortality in thyroidectomy is about 1% and in mildly toxic cases is very much less than that. Comparatively young patients with goitres giving a high metabolic rate will stand operation much better than will older patients with a much lower metabolism, but of long standing. Those patients who have had a nodular goitre for a long period of years even though their toxicity seems only moderate must be handled with great care. They are the ones in whom the heart reserve has been used up and who will not stand even a mild thyroid storm.



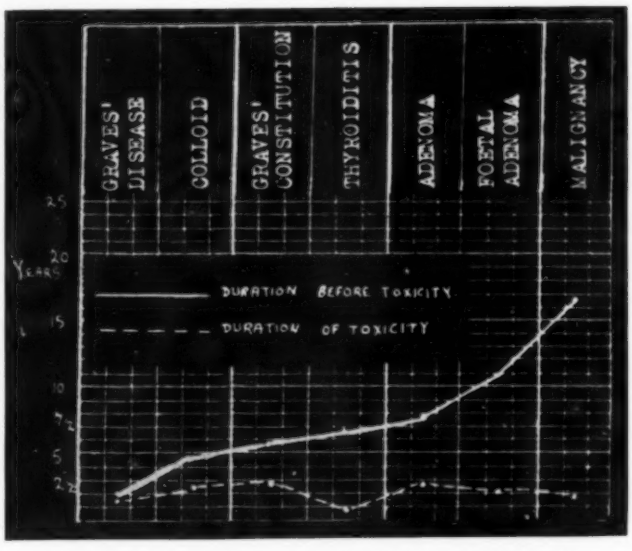
Distribution in years of pathological types

Exophthalmic goitre, which is a misnomer inasmuch as only about 60% of these patients suffering from it actually develop an exophalmos, is usually an acute disease. It always becomes toxic within a year or two after its initial appearance and its virulence increases until death ensues or treatment by medication or surgery is instituted.

The toxicity of this type of goitre is better controlled by the administration of iodine than in any other, but it can not be held in check by medication indefinitely or permanently except in occasional instances, unless the treatment is augmented by a marked reduction in the patient's normal activity. This type of goitre occurs at any age. It is rare in early youth, but does occur in very young children. My youngest patient of this kind was six years of age, but Grave's disease had been seen within the first year of his life. It is not endemic and may occur in any locality. Surgery should be instituted early in order to avoid the constitutional ravages of the disease which inevitably follow if the disease is left untreated. A survey of statistics offered by other clinics and also of my own cases indicates the necessity for



Comparison of average weights and durations of types of goitre



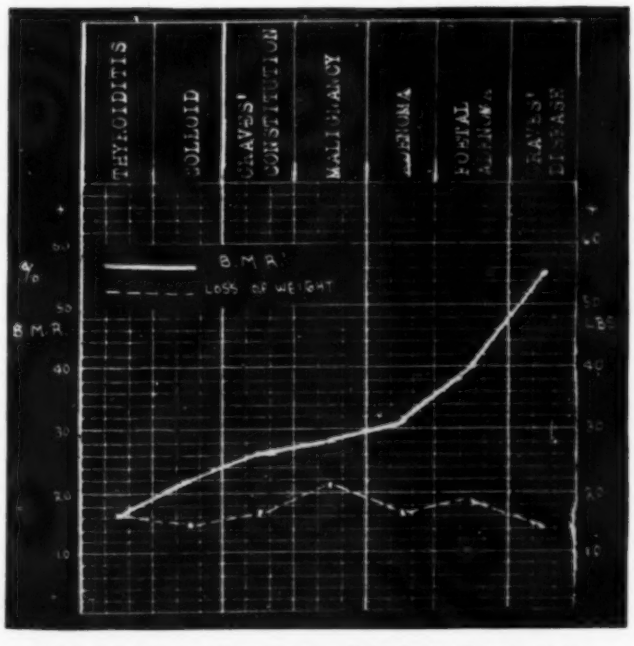
Contrast of duration of goitre before toxicity to duration of toxicity

secondary operations in at least 10% in these cases, and one case has been reported that was operated eight times within a few years.

Grave's constitution of Warthin appears to have much the same characteristics as exophthalmic goitre with regard to control by iodine and recurrence.

Undoubtedly both are constitutional diseases to a greater extent than are other types of goitre. The dangers of operation are somewhat lessened in them in that they are acute diseases and are apt to come to operation before their toxicity has destroyed the strength of the heart.

Riedel's struma, struma lymphomatosis of Hashimoto's disease, has a very hard feel. The thyroid may be only moderately enlarged and is very firmly attached to the trachea. It should only be operated when pressure symptoms appear. The thyroid cells have been replaced by fibrous structure to a great extent and the removal of any



Comparison of the average B.M.R. to the average loss in weight

of the thyroid gland only further reduces the amount of thyroxin secreted into the blood stream and will throw the patient into a myxoedema necessitating the administration of thyroxin for the remainder of the life of the patient. If the pressure becomes too great the thyroid isthmus can be split, relieving the constriction, but if there is no pressure it should be left to its own devices to avoid subsequent hypothyroidism.

Malignancy of the thyroid is as dangerous as in any other part of the body. In its early stages its presence is usually marked by a nodular enlargement which simulates adenoma of the thyroid, but the nodules may be small and difficult of detection. The signs and symptoms of goitre appear in various stages of intensity. Malignancy is seldom seen in simple hyperplastic or endemic goitre until such time as nodules appear. The patient may pay little attention to the enlargement or actually not realize that nodules are present until the malignancy has developed in them and started to break through the membrane or capsule. Four percent of my nodular goitres have been demonstrated to be malignant under most careful pathological examination. These have been undiagnosed for the most part as there is no method of distinguishing between a nodular goitre and a nodular malignancy. Even if there are no evidences or metastases or direct extension the papillary types are very apt to recur, probably from the few cells that are left after the sub-total thyroidectomy has been performed.

Unfortunately even at operation frequently the malignancy can not be detected and it is demonstrated after the laboratory has gone through the gland. It is then necessary to either re-open the neck and do a total thyroidectomy and then to administer deep x-ray therapy treatments or to rely entirely on deep x-ray therapy treatments to eradicate whatever cancer cells may still be present. When it is demonstrated that the carcinoma is of the adenomatous type and the operator is certain that all of the nodules have been removed x-ray treatments should be administered with the expectation that a cure will follow provided that direct extension through the capsules has not already taken place.

THE treatment of colloidal goitre in our hands has been moderately successful provided it was started early enough in life. Most of these parenchymatous goitres appear shortly after puberty and treatment should be continued from their first appearance until no further sign of enlargement is present. This may be over a period of from 10 to 15 years, and the administration of iodine at regular intervals during the years will keep the gland under control. After the patient has reached her middle twenties and if the goitre is still present further iodinization will be of little value. Usually the treatment is stopped too early. A very common history is for the patient to say that she had a goitre at the age of 15 or 16, which disappeared after a year of treatment, but that now that she has arrived at the age of 30 she again notices an enlargement of the neck which is nodu-

lar. Actually what has happened is that the goitre has never disappeared, but has been moderately quiescent and adenoma either diffuse or discrete has slowly developed.

In this colloidal type of goitre long continued treatment would have in most cases completely eradicated the disease. We have patients whom we have treated in this manner over many years in whom no signs of adenoma have ever made their appearance. Under no circumstances do we advise a thyroidectomy for simple goitre unless pressure signs develop, or the goitre has become so prominent that its appearance annoys the individual. The appearance of toxicity is evidence that adenomas have developed in the gland and these have become active. It is my belief that all adenomas should be removed. They all become toxic eventually. Before damage has been done to the heart and nervous system the operation is without much danger. The danger comes in waiting until the toxicity is well advanced and the heart reserve is dissipated. Except for some peculiar bizarre thing which may occur subsequent to any operative procedure a thyroidectomy at an early stage is as danger free as any other operation. Therefore as soon as an adenoma is evident, thyroidectomy is indicated.

In Grave's disease, which is acute and fulminating in character, we have a problem which is to some extent different from that of toxic adenoma. In quiescent adenoma the administration of iodine, as I have mentioned, may be attended with danger, but this is not true of Grave's disease. Iodine can be given in large and continuous doses over a long period of time. It may in certain instances control the virulence of the goitre. On the other hand the goitre usually continues to increase its toxicity in spite of the iodine, but this increase in toxicity is not the result of iodine administration. This is the type of goitre which will recur in from 5 to 10% of all cases, and the operation should be as nearly a complete thyroidectomy as possible without doing a total ablation. These patients will not be thrown into hypothyroidism if a few cells are left on the posterior capsule.

It must be noted that exophthalmos occurs in only about 60% of the cases of Grave's disease. Therefore, we may not rely on this diagnostic symptom alone for determination of the amount of thyroid structure to be removed. A better criterion is that of chronicity.

If the goitre is smooth and toxicity has appeared shortly after its appearance, it is probably of the hyperplastic type, and should be treated as such. Thyroidectomy is still the best treatment that we have for Grave's disease. There may be improvement at times during the course of the disease without operation, but a relapse almost inevitably follows which will be worse than the first attack. X-ray at times will give some moderate control, but will not cure. It should only be used in preparation for operation. Grave's constitution should be treated as Grave's disease. While it is not fulminating as Grave's disease, it is much more so than toxic adenoma. Fetal adenoma is of long duration and may be non-toxic for many years. As it is nodular in appearance it may not be distinguishable from simple adenoma and it should be removed when the adenoma first develops. The removal is not attendant with recurrence. Left alone it is prone to become malignant. It is interesting to note that goitre may be present for a varying period of time, before toxicity occurs. Frequently it is quiet for many years. I recently saw a woman 101 years of age with a large calcified goitre in her neck. From the history it had been moderately toxic rather early in life, had become calcified and for the last 50 years of her life had not been troublesome. A careful analysis of the histories of the patients coming to operation shows that after toxicity once occurs the patient will come to operation within a period of 2.5 years. That is to say that the patients will stand goitre for any length of time, but will not endure the nervousness and heart trouble caused by thyrotoxicosis more than an average of 30 months.

The size of the goitre has nothing to do with its toxicity. A large goitre filled with colloid or cystic in character may be relatively non-toxic, but a small goitre in which the cells have hypertrophied into the acini may be hardly palpable and yet evidence toxicity of the most virulent type.

I have brought to your notice this classification of goitre with the hope that by doing so some aid will be given in the treatment of these cases to those of us who are not specializing in the care of goitre cases, but who see them at more or less frequent intervals. The following questions are frequently asked me: "When should I give iodine with the hope of improving the patient, and when is it dangerous to administer?" "What cases should be operated, and what cases can safely be treated?" "How can I distinguish one type of goitre from another?" It is these questions in part that I have attempted to answer.

Evaluation of Head Lesions

LEOPOLD VACCARO, M.D., F.I.C.S.,
Former Chief Medical Adviser, Pennsylvania Department of Labor and Industry

SCALP—Ordinarily, lesions of the soft parts of the head are not very important from the standpoint of compensation unless they interfere with the function of the muscles of the face or cause definite pain and discomfort or reflex epilepsy.

Loss of hair following extensive scarring of the scalp, is subject to disfigurement awards. It is to be borne in mind that any operative trauma or injury to the nerve fibers, may result in alopecia. There may be localized neurotrophic alterations together with involvement of the sebaceous and follicular glands.

INJURY TO THE BONY STRUCTURE — Fractures of the skull are of a serious nature because they may involve the brain tissue or its coverings. It is not unusual for individuals having the bones badly traumatized to resume work without any after ef-

fects, especially where there are no depresesd fractures or osseous comminution. The fractures may be linear, single, multiple or comminuted or there may be bone ablation.

Compression may result from the presence of bony fragments, laceration of the meninges or lesion of the brain substance proper. Fistulas may result from the presence of foreign bodies, following explosion, sequestra, trephining of the skull or periostitis. Their importance depends on complications within the cranium which determine the prognosis.

Constitutional disease such as syphilis or tuberculosis may have an important bearing in the matter of localization, resulting in chronic osteitis of the vault following a trauma. Loss of osseous substance of the skull which is not amenable to a plastic operation and exposes the brain tissue to additional trauma, together with changes in the brain coverings, may result in headache and dizziness, preventing the worker from working in any hazardous occupations. While the symptomatology may be entirely subjective, it may be justified by the new anatomical deficiency and injury. Partial epilepsy and mental manifestations may follow. Partial disabilities of these bony breeches range from 50% to 100%. Where there is no loss of substance but only headaches, the disability may be computed on the basis of 20%. Where there is simply vertigo without falling, 25% loss of earning power may be considered.

Cases following trephining of the skull are extremely variable particularly if adjusted with a protective plate, although these subjects are unquestionably limited in their full activities, whether in the factory or ordinary life. They should be awarded no less than 30% to 50% partial disability.

Lesions of the Cranial Nerves — The cranial nerves may be directly damaged by a bony particle, stretched or compressed by a fracture; or they may be severed at their origin or compressed by a hematoma. The result is usually paralysis.

The nerves most frequently injured are the 8th, 7th, 6th, 4th and 3rd. The optic nerve is rarely injured. They may be directly contused or some destructive process may damage them at their root, course or point of exit. In the case of the auditory nerve, there may be damage in the labyrinthine terminations. This nerve is involved in injuries of the middle ear.

Optic neuritis may result from indirect fractures of the orbital parietes. In facial palsy when it appears immediately, the facial nerve is usually injured during its interosseous course. If the paralysis occurs a few days following the fracture, it may be ascribed to a blood collection which has subsequently organized or to callus formations of the fracture.

Facial paralysis may also have a central origin due to contusions of the brain proper, independent of any fracture. Facial palsy should be awarded from 30% to 40% partial disability and at least an additional 50 weeks for disfigurement. Paralysis of the 5th or trigeminal nerve should be compensated to the exten of 20% to 30%. The remaining four pairs of nerves; the 9th or glossopharyngeal, 10th or vagus, 11th or accessory and 12th or hypoglossal, produce a varied clinical phenomenology due to the particular paralysis involved. The various syndromes include paralysis of the vocal chord, of the tongue, the trapezius and the sternocleido-mastoid. Ordinarily, in compensation practice, such injuries are not common but they occur more frequently in war surgery. Awards for these last four nerves range from 30% to 40% partial disability.

Brain Injuries

Traumatic Cerebral Concussion, Contusion and Compression—In order correctly to evaluate head injuries in relation to the consequences, an accurate history must be obtained of the signs and symptoms immediately following the injury.

There are two clinical periods to be considered; the syndrome of shock and the manifestations of a period of consolidation which indicates exactly the nature of the organic lesions produced by the trauma.

The usual phenomena encountered in brain injuries are, concussion, contusion and compression of the brain.

The *concussion* has been limited to those cases which do not show the clinical syndrome of cerebral pressure. The mechanism of concussion is not, as yet, very clear. It is ordinarily conceived to result in a violent agitation of the cerebral mass whose cohesion and consistency is very slight. This jolting is usually caused by direct injuries of the head or by violent dislocation of the whole body.

Concussion is characterized essentially by immediate unconsciousness. The patient may be in a deep coma or can be aroused. The symptoms of shock such as rapid pulse, low blood pressure and general absence or diminution of reflexes, are present. Headache, vertigo, nausea and vomiting with partial or total loss of memory relating to the events of the injury, are often present. The severity and duration of the symptoms are most variable. In mild cases, recovery occurs after a brief interval. In more severe cases, the victim may be left for a time incapacitated both physically and mentally. In still more severe cases, profound unconsciousness may persist for hours and the reaction to external stimuli may be entirely abolished.

In the latter type of cases, there may be microscopic contusions and petechial hemorrhages. The mortality in concussion has been variously estimated from 3% to 5%. The morbidity often presents a picture of persistent headache, vertigo and the various types of mental disturbances which may last at least 10 days. Very few patients may retain these symptoms indefinitely and may become totally disabled. These cases formerly were classified as traumatic neuroses. Recently, the technic of ventriculography has been employed for both diagnosis and treatment. Spinal fluid withdrawals in 5 cc. amounts alternately injecting after each withdrawal of 5 cc. of air. From 80 to 100 cc.

of fluid is withdrawn and replaced by an equal amount of air so that the arachnoid spaces are completely filled. The patient is sent to the x-ray room without altering the position and horizontal and perpendicular plates are made, after oscillating the head thoroughly so that the air is carried through all parts of the arachnoid space. The plates taken immediately after insufflation, usually show the air blocked off from the region where the headache has been located. In most favorable cases, plates taken 48 hours after the insufflation of air show it to be evenly distributed through the subarachnoid space. Some authorities advise great care in selecting cases of lumbar air insufflation because of unnecessary suffering. This method is not entirely free from danger although it may be satisfactory otherwise.

Compression: This condition is the result of intracranial pressure. It is usually due to a hemorrhage and unless relieved, in itself, will produce rapid death. It is ordinarily associated with extensive contusion and laceration of the brain.

Practically all cerebral traumas are accompanied by an increase in intracranial tension compressing the cerebral vessels and resulting in anemia and death. There is increasing stupor and a definitely progressive increase in blood pressure and decrease in pulse rate. As the time of death approaches, there is stertorous and Cheyne-Stokes breathing. The temperature rises rapidly and reflexes disappear and the patient becomes relaxed. The blood pressure rapidly falls to zero and respiration ceases. Death in cerebral injuries is often the result of compression of the medulla and the vital centers contained therein. Frequent observations of the pulse rate, blood pressure and spinal manometer readings, are made. The mortality following acute head traumas has been variously estimated from 30% to 40%.

Contusion: From a purely clinical standpoint, contusion and concussion represent lesions of degree only and one cannot be present without some degree of the other. Contusion, however, tends to be progressive and to become localized. There are numerous petechial hemorrhages in contusion observed at postmortem examinations. There may be lacerations as well. The spinal fluid will usually be blood stained.

There are other conditions from which brain traumas must be differentiated such as apoplexy, hysteria, alcoholism, diabetic or uremic coma. In apoplexy, there is immediate hemoplegia and the face is distorted whereas in concussion, it is pallid. Furthermore, the loss of consciousness is not immediate in apoplexy as in the traumatic coma. In alcoholic coma there is the characteristic odor. In hysteria, the symptoms are obviously psychic. Coma due to uremia, is generally preceded by convulsions and is associated with subnormal temperature and Cheyne-Stokes respirations. The reflexes are usually exaggerated.

There may be other conditions, such as meningitis, acute poisoning from carbon monoxide or advanced syphilis but ordinarily, a close clinical scrutiny will make the diagnosis clear. Traumatic shock is often associated with concussion as a consequence of head traumas.

Head traumas terminating fatally or in recovery, are frequently brought before compensation courts and a true picture is often distorted by the parties at interest requiring the unbiased opinion of an impartial surgeon for a true evaluation.

A case recently treated by me presented a diagnostic problem and is illustrative of the symptomatology of concussion, contusion and compression of the brain with a clinical picture of apoplexy in the absence of a definite traumatic factor. The patient was a white male, 57 years old, plethoric and addicted to alcohol. The history given was that on a Sunday night he was brought home in a state of intoxication. He was put to bed and seen by two general practitioners and a consultant who made a diagnosis of cerebral apoplexy on the basis of immediate hemiplegia and a distortion of the face, together with a semi-comatose state. The patient had always been healthy and his alcoholism was not doubted.

I saw this patient three days after the cerebral accident but could find no evidence of a head injury. He was very restless, the left pupil was dilated and his tongue was protruding to the left. He was partially irrational, the pulse was 64, respirations 22 and the temperature 98°. I transferred him to the hospital where a Roentgen examination of the skull revealed a fracture of the right side starting in the occipital bone near the margin with the parietal bone, passing through the lambdoidal suture and across the parietal bone. On that day about midnight, his temperature rose to 99°, pulse to 140 and respirations to 40. By the second day, temperature had subsided to 97.4°, remained stationary and the respirations were 28. He had muscular twitchings of the left side and a distended abdomen. Soon after, he developed twitchings of the entire body every three to four minutes which increased in frequency to every two to three minutes with a weak pulse. This was in direct contrast to the previous eight hours when he felt well and was calm and rather happy at being hospitalized. The twitchings continued the following day and became typical Jacksonian seizures involving the entire left side of the body. He was given 5 gr. of sodium luminal intravenously followed by 5 gr. sodium amythal three hours later, also by the intravenous route, and a 10 cc. ampule of 50% glucose for dehydration. The condition progressed from bad to worse and an infusion of 10% glucose in 500 cc. was administered. The blood pressure and respirations were taken hourly. The blood pressure was never uniform but oscillated from 110/90 to 98/66 while the pulse rapidly rose from 64 to 140; respirations to 46 and the temperature rose to 102°. The pulse became gradually imperceptible. He developed Cheyne-Stokes respirations and died.

Death occurred exactly seven days after the cerebral accident. The spinal fluid was almost pure blood. On admission to the hospital, the RBC were 4,700,000, the WRC were 15,000 and HG 95%, the

polys 75% and the sl 25%. An autopsy performed disclosed a large extradural hemorrhage with numerous hemorrhages in the brain substance. Further investigation by the detective bureau disclosed that this man had been struck on the face in a brawl and had fallen, striking the curb. The culprit was held without bail.

This case again demonstrates the correctness of the introductory statement that no head injury can be properly evaluated and diagnosed in relation to its sequences unless an accurate history is obtained and the signs and symptoms immediately following the injury carefully observed. The physicians who examined this man previously, did not have a history of trauma neither did they have the cranial roentgenogram. Apoplexy, alcoholism, concussion, contusion, and compression can be easily confused without the proper laboratory and x-ray studies and without a correct history of the injury.

Intracranial Hemorrhages

INTRACRANICAL HEMORRHAGES are ordinarily associated with the alcoholic or syphilitic vascular diseases which render vessels prone to spontaneous rupture and a generalized arteriosclerotic hypertension. Traumatic intracranial hemorrhages may be extradural, intradural, intracerebral or intraventricular. The extradural hemorrhage occurs in the temporal region and is due to rupture of the middle meningeal artery as the result of a cross line or depressed fracture. This artery grooves the bone from the foramen spinosum to the longitudinal sinus. The squamous portion of the temporal bone is most frequently injured. When the middle meningeal artery is ruptured, the dura is gradually stripped from the bone and its many branches are successively torn. This increases the original hematoma. It is estimated that this artery is torn in 90% of the serious cranial injuries.

The symptoms of compression arise gradually in direct ratio to the amount of bleeding. Compression develops gradually and is recognized by localized symptoms and as the cranial capacity is reduced by this continued hemorrhage, general symptoms appear. There is at first, restlessness followed by coma. If there is instantaneous loss of consciousness, this is due to cerebral edema. There is a free interval of consciousness. This lapse of time takes place between the external injury to the head and the appearance of the general symptoms from compression, particularly coma. The shorter the interval, the greater the danger. This period may last several hours or several days during which time there is no clear-cut clinical picture except, perhaps, slight headache.

At times, the newly formed blood clot may so compress the middle meningeal artery as to actually arrest bleeding. Again, this coagulum may become detached and the hemorrhage resumed.

Besides restlessness and coma as previously alluded to, the behavior of the pulse is very important. It is slow, full and bounding. When its rate and force are altered, there is distinctive danger. As the intracranial hemorrhage increases, the pulse becomes more rapid and more thready with a drop in blood pressure. A similar course is noted by the respiratory curve. When the intracranial pressure is under control, the respirations are slow and regular. When compensation breaks, they become labored, shallow and irregular and the Cheyne-Stokes respiration eventually appears indicating a very grave prognosis. The behavior of the temperature curve is also important. As long as it remains normal, or below 102°, it is relatively safe but as it rises, it indicates a bad outlook.

Vomiting of frequent repetition and explosiveness, points to marked intracranial pressure and is found in the earlier stages of compression. Papilloedema or enlargement of the veins, with haziness on the inner margin of the disc, appears after a day or two.

As the blood is reabsorbed, there is a diminution of internal pressure and therefore a reduction of the papilloedema. At any rate, an ophthalmoscopic examination should be made.

Of all the signs of localized injuries to the brain, the most important is hemiplegia. It may follow direct injuries, extradural hemorrhages, contusion of the brain substance or intracerebral hemorrhage or edema. The resulting paralysis is in direct ratio to the intracranial pressure. A depressed fracture does not necessarily result in high intracranial pressure, depending largely on the extent of injury to the brain substances.

Extradural or subdural hemorrhage may be assumed when the paralysis appears first in the face, then arm and later the leg. A direct contusion or intracerebral hemorrhage usually results in paralysis of the entire side.

There is also progressive loss of sensory function from face to arm, to leg. The convulsions resulting from intracranial pressure are usually Jacksonian in type when the motor area is involved.

When other lobes are affected, the convulsions are less frequent and less characteristic. When the deep reflexes are exaggeratd, the cerebral lesion is assumed to be on the side opposite to the hemiplegia.

A positive Babinski test bilaterally indicates a lesion to the brain stem or a lesion involving both motor tracks of the brain.

The injuries to the cranial nerves have previously been covered. There may be loss of smell, blindness, deafness or other disturbances pertaining to the particular function of the involved cranial nerves.

Cases of cerebral occlusion or thrombosis with a distinctive history of head trauma or contracoupe, often come before compensation courts for ajudication. The lay referee is naturally bewildered by the conflicting medical testimony presented by the plaintiff and defendant. The contradictory medical opinions further confuse an already complex and puzzling picture of head lesions.

One of my recent cases concerns a barge captain, 60 years old, who gave a history of being struck on the right side by an iron bar while winding a hawser around a capstan. He suddenly experienced a terrific roaring in his head and shortly thereafter noticed a weakness of the right arm and leg. He held on with the left hand to keep from falling. Next he seized a plank and laid it on the barge extending it to the dock. As he jumped three feet from the barge to the dock, he collapsed and became aphasic and was brought into the accident ward with a flaccid paralysis of the right side of the body and face. The reflexes were weak bilaterally, the arteries firm and the blood pressure 170/90. It was not determined how soon after the jump, he developed hemiplegia and whether or not it was accompanied by loss of consciousness. It was stated that two days prior to the occurrence of this vascular accident, he felt dizzy and early in the morning, or about four hours prior to the occurrence of this vascular crisis, he felt sick.

A neurological examination revealed that he was definitely aphasic with both receptive and expressive difficulties. He was irrational, expressing himself and showing evidence of mental confusion. He did not appear anxious or depressed and was almost elated. There was no marked rigidity or Kernig's sign. The fundi were visualized and showed only sclerosis. The pupils were irregular and responded moderately to light. He had a right central facial palsy and deviation of the tongue to the right. No other cranial nerve abnormalities were noted. The right abdominal reflexes were unattainable. He had a complete flaccid paralysis of the right arm and leg; the tendon reflexes were bilaterally diminished more so on the left than on the right. There was a good Babinski sign on the right and a suggestion of right hemi-anesthesia to pin-prick.

The diagnosis was made as occlusion of the branch of the left sylvian artery accompanied by considerable edema of the brain or considerable vascular changes in the right hemisphere of the brain. The etiological diagnosis was vascular arteriosclerotic or luetic. The Kolmer complement fixation—reaction, the Wassermann and the spinal colloidal gold reactions were negative. The spinal tap showed a pressure of 120-170 mm. of water. The first tube of 1 cc. was clear. The second tube of 2 cc. was also clear. The third tube would not flow, so 3 cc. were aspirated and found to be bloody. An additional 1 cc. was allowed to flow out by artificial pressure and was less blood tinged than the first tube.

Two weeks after this cerebral accident the patient had definitely improved and his speech had become almost intelligible. He was discharged one month after admission, at which time his vocal center worked fairly well and he appeared quite affable and cooperative. His blood pressure was 144/70 on discharge, and he had a systolic murmur and hemiplegia.

The question of cerebral hemorrhage and ensuing hemiplegia due to trauma on the right side of the body, was strongly emphasized by the claimant's attorney. When it was proved that the patient did not have any head lesion on the left side, the question of a sudden rise of blood pressure, due to a possible, but not proved, fall, was advanced as the cause for rupture of the sylvian artery.

This case did not present an alcoholic or syphilitic vascular disease but did have a generalized arteriosclerosis and hypertension. There was no history of immediate unconsciousness, and the symptoms developed gradually pointing very clearly to thrombosis or occlusion of the left sylvian artery.

This case is an example of the medical legal riddles which the medical expert is often called to elucidate and evaluate for the compensation court.

Traumatic Abscess

IT IS estimated that about 50% of the brain abscesses are due to trauma. They may result from penetrating wounds such as gun-shot lesions, skull fractures, and fissures of the cranium. Trauma without a solution of the continuity of tissue, may give rise to an intracranial abscess. Rupture of the tympanum, otitis media, or mastoid disease may be frequently a cause.

The softening of the brain tissue following occlusion of a cerebral blood vessel, offers a favorable field for a pre-existing or secondary infection. A blow on the head may be followed with an abscess a few weeks or months after.

The traumatic origin of an abscess should not be difficult to determine if there is a history of laceration of the scalp or a fracture, but it is often impossible to make a correct diagnosis because in the beginning, the symptoms preceding localization and compression, are not very clear. The site of the trauma and the absence of other non-traumatic causes, should be taken into account. Abscesses are frequent in patients suffering from middle ear diseases followed by thrombosis of the lateral sinus. An abscess may be circumscribed and encapsulated. Ordinarily it develops slowly and without symptoms and, often, a long time after a head wound has healed. It is hard to differentiate it from meningitis from which it is often derived either by direct continuity or through the blood stream.

One of my cases was a man 33 years of age, an electrician by occupation. One night he was asked to examine a grinding machine which was out of commission. He opened the coil box and claimed to have been affected by its peculiar odor. Ten feet away he collapsed and was found unconscious. He was immediately taken to a hospital and the following morning was discharged. An x-ray taken of the skull showed no evidence of fracture or structural intracranial changes. He was again seen three days after, when he showed a change in personality and mental confusion. One week later, he became comatose and was admitted to the hospital for study. On admission, he appeared in a somewhat

semi-comatose state, slightly restless, pallid and cyanotic in the lips. He could be aroused with effort and mumbled about a headache of three days duration which was never experienced before. His record as a worker was excellent; he had worked for a number of years without any lost time. The pupils were equal and reacted normally to light and accommodation. The blood pressure was 136/78. The reflexes were normal but the pulse was slow, very much like a cerebral pulse. The heart, lungs, abdomen and extremities were negative. The blood count showed 14,800 leucocytes, RBC 5,000,000 polys 88%, lympho 69%, mono 37%; blood sugar 86, urea 14, creatinin 1.4; urine negative. A cardiograph showed sinus bradycardia with no conduction defect and no axis deviation. The spinal fluid was clear with 500 mm. of pressure. There was a slight increase in sugar reduction. There was a white cell count of 50 and a plus globulin. There was no history of ever having any apoplectic seizures or any similar spells. The blood Wassermann and Kahn were negative.

On the second day he refused to respond and was still semi-conscious. He had a beginning catatonic stupor involving the muscles below the axilla and the shoulder region. The upper extremities below the axilla were not involved. He had mild muscular twitchings with slight movements of the extremities more marked on the right side. The right pupil was about twice the size of the left. He exhibited definite convulsive seizures, was unconscious and had respiratory difficulty. The blood pressure dropped to 120/86, the pulse was irregular. He expired on the seventh day after admission to the hospital.

In this case the neurological and eye ground status were negative. Autopsy revealed a horseshoe abscess involving the temporal and parietal lobes with a thick inflammatory capsule and yellow-greenish pus. Since the abscess involved the silent region of the brain, no definite prenecropsy diagnosis of the brain abscess had been possible. His temperature had been constantly subnormal.

The relationship between the fall and the subsequent death could not be definitely established; neither could any other cause be determined since a complete physical examination, including the ear, showed no abnormalities.

Traumatic Meningitis

THIS may be caused by a hematoma following a simple contusion, infection reaching it by the blood stream or by direct continuity. It may be secondarily due to an osseous necrosis of the damaged cranial plates. Meningitis can also follow a contusion at a distant point which has been subsequently infected and the germ carried to the meninges by the hematogenous route.

If the meningitis is tubercular it may have lit up an old focus which was either calcified or caseated. In this case, trauma is considered to be directly responsible for the aggravation of a pre-existing condition.

Injection Treatment of Hernia

FREDERICK W. SLOBE, M.D., F.A.C.S.,
Chicago

THE objectives sought for in curing a hernia are the eradication of the sac and strengthening of the wall. Surgery has the distinct advantage in that the field is laid bare exposing the pathologic anatomy and allowing, after the excision of the sac, the use of whatever method is indicated to strengthen the wall. The injection method offers as a substitute either an agglutination of the walls of the sac or a fibrous constriction of the tissues surrounding the sac especially at its neck. Neither in obliterating the sac nor in strengthening the wall can the injection method approximate the results obtained by direct surgical attack.

The injection method, however, has definite advantages when viewed from the standpoint of mortality and complications. The surgical mortality is about 1% whereas the mortality by the injection method is so low that it is almost negligible. The complications inherent to any operation are singularly absent in injection therapy, serious complications being exceedingly rare, assuming, of course, that the correct technique is used.

The injection method, moreover, has a greater range of applicability to the profession because although requiring skill, training, and care, it can be used by a far greater number of physicians than the operative method because it is essentially a much simpler procedure.

There are many contra-indications to the injection method but it can be used in many instances where surgery is contra-indicated as in the aged and infirm or in those who have serious constitutional ailments. Its use as a palliative measure with truss-wearing merits emphasis. The treatment of individuals with relaxed rings and potential hernias constitutes a field for the injection method which has many potentialities not yet adequately explored. In elderly men the frequency of flare-ups following postoperative catheterization in latent prostatics, followed by definite clinical prostatism suggests caution as to routine operation in this age group. Truss-wearing plus injections may be preferable here.

The high recurrence rate of the injection method constitutes a very serious drawback. The writer feels that if the truss is worn only six months the vast majority of the patients will have sufficient absorption of the fibrous tissue so that a beginning recurrence will take place although the patient may not be aware of it until later. The only way a high recurrence rate can be avoided is by wearing the truss indefinitely together with periodic re-injections.

This constitutes a very definite disadvantage because most individuals do not like to wear a truss indefinitely.

DR. SLOBE'S summary of a presentation given at a meeting of the MICHIGAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, November, 1940.

Most qualified observers of experience with the injection method have found that their initial optimism is very markedly tempered by the passing of time and as a result they are narrowing their indications for its use. This indicates that the present trend is reducing its use as a curative measure but increasing its use as a palliative measure.

From the standpoint of local results surgery remains the method of choice but the injection method has certain attractive features when used according to its restricted indications.

The Nurse in Industry

—*Relationship to the Physician*—

STANLEY J. SEEGER, M.D.,
Milwaukee, Wisconsin

THE industrial nurse is part of the health and safety program of industry and her duties include all nursing services which promote the health of workers. These are not only the usual first aid measures and nursing care, but also the important duties connected with the control of those factors which cause illness, fatigue and reduced productivity. In discussing the nurse in industry there is need for a concept of her position which is broader than that usually held. Most people think of industrial nursing as confined to first aid in large manufacturing plants, but surveys such as that made by HELEN LEHMAN in Cleveland show that this is not correct. Some of the best work of the industrial nurse is done in organizations where there is no manufacturing.

In giving consideration to the problem of physicians and nurses in industry it must be realized at the outset that, contrary to the usual belief, most workers are employed in small plants. A majority of workers, 80%, work in groups of less than 100, and 97% work in groups of less than 250. Industrial physicians may be classified as those who are full-time in one plant, those who are on part-time in one or more plants, and those who are on call.

This grouping may also be applied to nurses. It becomes apparent that the difficulties of establishing relationships and of coordinating activities will reside in those activities which are centered about the small plant rather than those of the large manufacturing establishment. In large plants with full-time medical direction the duties of the nurse are largely clinical duties and the scheme of organization of the establishment will define her ethical relationships with physicians. In the small plant the nurse will find that a restriction of her duties to the clinical type of service will not justify her employment, and the necessity of well understood and well defined medical contacts and supervision is most needed in this particular field. If the physician's concept of industrial nursing is based purely on ideas of clinical nursing obtained in hospital practice, the nurse will find it difficult to fulfill all of the possibilities in the industrial field. In order properly to develop the possibilities of nursing services in industry, the nurse should be trained in sanitary inspection and supervision and employment activities, in the maintenance of legal requirements of health control, in safety supervision, in recreational programs and in the safeguarding of conditions connected with eating facilities. Her value in the small plant is proportional to her ability to take responsibility for other than usual clinical activities.

This definition of the place of the nurse in industry is somewhat extended because it serves as a basis for discussing her relationships with the physician. Because of the fact that nurses are often employed through non-medical personnel management in plants, the medical contacts are, in many instances, of a casual nature. The necessity of establishing the proper liason between the medical profession and the large body of nurses now practicing in small industry with relatively little medical supervision is obvious. As this field of nursing endeavor develops, the need for medical supervision will become more and more apparent. As is true throughout industrial medical practice, the advances of scientific and industrial processes have, in many instances, outstripped the organization which we have perfected to make this knowledge of practical value to the workers. Through programs such as this, various professions concerned with the health of workers have an opportunity to present the problems with which they must deal, and gain from the discussion a more satisfactory answer to the problems of medical control of nursing services in the small industry.

THE National Organization of Public Health Nursing has outlined the qualifications of the industrial nurse in a most satisfactory manner. The problem of training nurses to meet the requirements demanded in this exacting field has been the object of study of various groups including the National Organization of Public Health Nursing and the Council on Industrial Health of the American Medical Association. Schools of nursing should be brought to emphasize the possibilities in this field for the properly qualified nurse. Training in industrial health nursing, as in public health nursing, should be developed on the graduate level. The question has been raised as to whether it is advisable to establish training centers for nurses in this field before a demand arises. The argument which ensues is quite similar to that regarding the chicken and the egg. It is argued by some training centers that they do not wish to give training because they cannot assure their graduates of positions when they have completed their courses. On the other hand, the employers have had the experience that the capacities of the usual nurse are limited and unless a campaign of education can be carried out informing employers of the

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position which the nurse can assume, no great demand for individuals with this type of training can be expected. Industry, in plants above the size of the smallest, needs some type of nursing activity, and nothing would do more to promote the development of a demand than the existence of a group well trained in this field.

The answer to the problems of the small plant will probably be found through part-time nursing services. One promising development in this field is the interest of the Visiting Nurses' Associations throughout the country in the possibility of extending their activities in public health to the industrial field. County medical societies and state medical societies in the industrial areas, through their committees on industrial health, should carry on the necessary contact and supervision of industrial nursing activities. With the emphasis which has been placed on the worker in the defense program, this type of health activity is particularly important, and the nursing profession can make a most valuable contribution by promoting the necessary activities. The community of interest between the nursing profession and the medical profession has long been recognized and it is unfortunate that the present methods of coordinating the activities of physicians and nurses are as haphazard as they appear to be.

In 1932 a special committee of the State Medical Society of Wisconsin prepared a list of suggestions for the guidance of the nurse in industry which was very largely an outline of first aid measures. At the time of its first publication the foreword stated that, "The nurse has many opportunities for splendid service. She is a nurse, an educator and the first line of service to the injured or sick employee. Her services, however, must not exceed those which by training and license she is prepared to render." It is recognized that the industrial health nurse in the past has not generally had the opportunity or the preparation for building a broad program of prevention in education and this need has been apparent to leaders both in medicine and in nursing. The national preparedness program has given great impetus to the entire field of industrial health work. Industrial nursing, as industrial medicine, will, as a result, experience rapid development in the next few years. The Council on Industrial Health has recognized the desirability of encouraging efforts to improve the professional standing of the industrial nurse. Current projects being undertaken by nurses themselves are (1) the definition and establishment of suitable training courses; (2) the preparation of a handbook on industrial nursing, and, (3) an investigation of the possibilities of employment of industrial nurses as the first step to providing preventive nursing practice to the small plant. It is important for Wisconsin physicians and nurses to realize that their state, through Miss Joanna Johnson, chairman of the committee on industrial nursing of the National Organization of Public Health Nursing, has assumed an important part in the promotion of this program.

Prevention of Industrial Dermatitis

M. J. Reuter, M.D.,
Milwaukee, Wisconsin

DISEASES of the skin due to occupation represent a considerable percentage of all cutaneous disease. Schwartz, of the U. S. Public Health Service, estimates that 1% of the industrial workers in the United States are annually affected with occupational dermatoses. Because it is impossible in one short paper to discuss the prevention of all skin diseases arising as a result of occupation, I shall limit my discussion to the most common, namely industrial dermatitis.

Industrial Dermatitis

INDUSTRIAL dermatitis may be defined as "an inflammatory condition of the skin incidental to systematic work or labor or habitual employment." Industrial dermatitis may be due to a substance which will produce an inflammation on contact with any individual's skin. This substance is termed a primary irritant and the inflammation, primary irritant contact dermatitis. Industrial dermatitis may be due to skin sensitivity of the worker to a particular substance, which would not produce inflammation on a normal individual's skin. When this occurs the condition is called sensitization contact dermatitis.

It is almost axiomatic that any substance must wet the skin or be soluble or mixed with the secretions of the skin, such as the sweat, in order to produce inflammation. By far the great majority of cases of industrial dermatitis are due to chemicals that are primary irritants. The first contact with the irritant in sufficient concentration or prolonged contact with weaker dilutions, is capable of setting up a dermatitis. In dermatitis due to specific skin sensitivity, a period of exposure is necessary, during which time the substance is handled with impunity. This period may be one of several days or months or years. After sensitization develops, the worker reacts to a dilution of the substance which would have no effect on a normal or unsensitized person.

Prevention of Dermatitis due to Primary Irritants

THE common primary irritants contacted by workers are acids, alkalis, hypertonic solutions, metallic salts, soaps, gases, vapors, cement, plaster, lye and physical agents. The degree of inflammation depends on the substance itself, the concentration and length of exposure. Persons with thin blonde skin or with dry skin are more readily affected. Dermatitis due to primary irritants is much more prevalent in the winter time because the oil and sweat glands are less active and thus the protective action of these secretions is lost and because the humidity is low in factories and homes, which further tends to a dry skin.

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There is little excuse for the development of dermatitis due to primary irritants because mechanical means of protection and proper education of the worker will prevent this type of skin reaction. Irritating fumes and dusts can be removed at their source by properly installed suction hoods and adequate ventilation, which is essentially an engineering problem but has proved its value in many industries. Protection of the skin can be afforded by the use of acid-resisting and alkali-resisting gloves, aprons and shields. In certain cases impervious protective pastes applied to the skin before work are often of great value. Clean protective clothing and adequate facilities for cleansing the exposed parts should be available. However, it may instances a primary irritant dermatitis is due to agents used to cleanse the skin during and after work rather than by substances handled in the course of the daily job. Horner has remarked that he had observed more cases in which dermatitis was due to the improper use of cleansing agents than cases in which the dermatitis was due to contact in the occupation. In Downing's series of 2,000 cases of skin disease arising in industry, in 249 soap and water constituted a contributory factor. Later I shall discuss protective medicinal applications to the skin and harmful and harmless methods of cleansing the skin.

While simply primary irritant dermatitis as a rule responds readily to proper treatment, it should never be passed over lightly because the worker's skin may become sensitized to the primary irritant, leading to a complex, severe, disabling, sensitization dermatitis which may continue for months and necessitate the individual giving up his particular line of work. Again, any skin that is the seat of inflammation from one agent may acquire sensitivities to other agents.

Sensitization Dermatitis

IT IS fairly well agreed that almost any chemical compound can produce sensitization dermatitis. With the rapid advance of chemical research the worker is constantly being exposed to numerous new sensitizing agents not only at work, but at home, even to the extent of the clothes he wears. Recently, there has been quite an epidemic of dermatitis from stockings and men's shorts due to a synthetic resin used as a finishing agent on the garments. In sensitization dermatitis, the areas of inflammation are not only the exposed parts that contact the agent but often the greater share of the skin surface is involved in the process. This may mean weeks or months of disability and frequently a change in occupation. Once this process has developed, the skin becomes sensitized not only to the original agent, but also to many substances, at work and at home.

Some persons become sensitized more easily than others, many with the first contact, others only after weeks, months or years of repeated exposures. Strong concentrations sensitize more readily than weak. The greater the exposure, the more likely the sensitization. Once sensitivity develops it may remain present for years, in some perhaps, it may disappear with time.

Osborne has stated that the problem of preventing sensitization dermatitis primarily resolves itself into four phases: (1) the proper selection of the workers; (2) the elimination of unnecessary hazards; (3) the prevention of exposure to sensitizing agents; and (4) the early recognition of dermatitis.

Selection of Workmen

REGARDING the selection of workmen, one type of skin is as easily sensitized as another. A dry skin is desirable in industries where gases and dusts are present. Where liquid irritants are employed, a moist skin is desirable as it protects the skin and dilutes the irritant. The pre-employment performance of patch tests is of no practical value and may, in fact, sensitize the applicant's skin. Persons with a history of eczema or the presence of a contact dermatitis are more liable to acquire other sensitivities than a normal person and should not be employed in industries using materials known to produce a dermatitis.

Elimination of Unnecessary Hazards

THE elimination of unnecessary hazards by industry offers much in the prevention of dermatitis. Chemical compounds known or discovered to sensitize the skin can be eliminated in favor of less harmful compounds. This requires cooperation between the dermatologist, to discover the offending agent, and the industrial chemist, to substitute a less harmful compound. Before a new chemical is introduced in industry, consideration should be given to its effect on the workmen and if found to be a harmful agent, adequate precaution should be taken to protect the worker or a new compound substituted.

Prevention of Exposure to Sensitizing Agents

PREVENTION of exposure to sensitizing agents may be accomplished by the use of closed methods of manufacture, by employment of suitable exhaust hoods to carry off gases, fumes and dusts, by proper cleansing facilities, and by instruction to workers regarding the danger of their occupation. Chemically resistant aprons and gloves, supplying the maximum degree of protection, should be insisted upon being worn by all workers handling dangerous substances. Complete change of clothing before leaving the plant should be insisted on. Often a double set of locker rooms, one for work clothes, the other for street clothes, would be highly desirable. Shower baths and sinks should be available and used to permit removal of possible offending agents from the skin as soon as contacted.

The early recognition of sensitization dermatitis is important because one or two weeks delay may mean the difference between a dermatitis lasting for a few weeks to one requiring two or three years to subside. When one worker develops a dermatitis, close observation of other workers

handling the same substances should be a routine. Workers should be educated to report promptly any skin eruption. Once a true sensitization dermatitis has developed the worker should be sent home and remain there under treatment until well. On return to work he should be placed on a dry job for some weeks. Later he may be permitted to work with the substance which produced his original dermatitis with instructions to report immediately any recurrence of his trouble and then promptly be taken off the job. The chances of the average person who has contracted a dermatitis having a recurrence, provided he continues at his same occupation and under the same conditions, appear to be about fifty-fifty with the edge slightly in favor of his suffering a recurrence.

Regarding the first aid treatment of industrial dermatitis, in my opinion bland, soothing, wet dressings, lotions or ointments should be the only medicaments permitted. As standard time honored remedies, I would include boric acid wet dressings, calamine lotion and zinc oxide ointment.

Protective Applications

IN THE prevention of industrial dermatoses protective medicinal applications, in the absence of mechanical measures of protection and when gloves are not worn, offer some degree of protection. There are a number of protective creams, liquids, ointments and pastes available commercially in this country. The exact ingredients of most of them are not known. The majority apparently contain paraffin, wax, gum, sodium silicate or casein which leaves a film on the skin. Klauder has recently published a number of different formulas as a protection to the skin under varying working conditions. One of the best types of protectives he recommends is a thick paste which leaves a water-proof impervious covering on the skin. It consists of one part zinc oxide, one part kaolin and two parts white vaseline. Its disadvantages are that it is somewhat sticky and it soils objects in contact with it. Therefore, it is appropriate only in certain work. The following formula I have found to be of some value in the prevention of dermatitis in female hosiery workers handling silk wet with a highly alkaline solution. It consists of:

Lanoline refined	9.00
Stearic acid	50.00
Triethanolamine	2.70
Carbitol	18.00
Sat. sol acid boric	120.00

This makes an ointment of the vanishing cream type which serves to protect the skin from the alkaline solution.

A greasy preparation could not be used, as this would leave a non-removable stain which would show on the finished stockings.

In the prevention of dermatoses in those who work with petroleum and lubricating oils, Scott recommends crude castor oil, since it is impervious to paraffin. Downing advises a greaseless cream and equal parts of boric acid solution and alcohol before and after work. Workmen whose hands become extremely soiled should apply some oil before work as this greatly facilitates the removal of dirt and grease by the use of soap and water after work.

Cleansing the Skin

AS PREVIOUSLY stated, the important role cleansing agents play as causative factors in industrial dermatitis has been stressed by a number of writers. Cleansing agents such as strongly alkaline soaps, fat solvents such as gasoline and turpentine, are frequent causes of primary irritant dermatitis. It is a common practice among industrial workers to use on the hands for removal of grease, dye, paint or other stains, solvents or chemicals that are used in their work. Some of the agents employed are alcohol, naphtha, acetone, amyl acetate, kerosene, benzene and various acids and alkalis. Most of these chemicals are not only primary irritants and drying to the skin but also some are sensitizing agents. Workers in paint, varnish or lacquer invariably use turpentine or paint thinners to cleanse the skin. Garage mechanics use gasoline; machinists, lithographers and printers use kerosene. These practices are bad and every effort should be made by the plant personnel to curb them.

A harmless method for the removal of dye or other stain from the skin is the application of a 10 to 30% solution of sodium bisulfite ($Na\ HSO_3$). To remove paints, oils and grease, a 1% to 2% solution of sodium silicate or 0.5 to 1% solution of sodium metasilicate can be employed. The application of a bland oil, (olive, cottonseed oil and liquid petroleum) has long been advised in order to facilitate the removal of dirt prior to the use of soap and water.

Another common harmful practice among industrial workers is the use of an abrasive soap and of a hand brush both of which are mechanically irritating to the skin and are not desirable cleansing agents and should be prohibited. Klauder advises as a substitute for the popular mechanic abrasive soaps, the following formula: Equal parts of sulfonated neat's foot oil and mineral oil containing 25% gelatine are added to white granulated corn meal in the proportion of one and a half by weight of corn meal to one part by weight of the oil mixture. To this is added 0.5% chlorbutanol to prevent growth of mold or bacteria. This substitute cleans soiled hands as effectively as does an abrasive soap and also furnishes some lubrication. Industrial nurses would do well to instruct workmen regarding lubricating the skin, especially in the winter time. Lanolin, olive oil, cottonseed oil, cold cream are all useful.

Soap

ONE is frequently asked the question, "what is a good soap?" This is not an easy question to answer. I frequently reply, "a soap made by a reliable manufacturer, and like a good cigar, costing not more than five cents a bar." The Federal government has laid down certain specifications for a good toilet soap which I shall not bother you with, except to say that much latitude is allowed in the manufacture of soap. Recent experiments

by Blank seem to show that cocoanut oil constitutes the principle irritating agent in soaps, and this oil is used in most toilet soaps. The term castile soap, formerly implied that pure olive oil was the sole fatty ingredient of the soap, but this is no longer the case due to ruling of the courts. Regarding soap in general, the soap less likely to irritate the skin is a toilet cake soap. Soap produces a dermatitis probably by its drying or defatting action on the skin and partly by its alkaline reaction. Soap powders are the most harmful because they contain great amounts of alkaline salts.

Sulfonated oils have recently been introduced to replace soap, they cleanse well but are occasionally irritating.

Conclusions

INDUSTRIAL dermatitis, then, is the most common skin disease in industry and is, to a large extent preventable. Preventive measures include proper selection of workers, elimination of unnecessary hazards, mechanical means of protection, prevention of exposure to dangerous chemicals, the early recognition and proper treatment of dermatitis and the education of the worker with particular reference to the use of proper cleansing agents.

As Nelson has so aptly stated, in the field of industrial health "Tell it to the marines" has its equivalent in "Tell it to the nurse." Perhaps no physician ever made a speech, however brief, before industrial nurses which did not begin, or end, or both, with the observation that the nurse, because of her intimate contacts with workers is the logical person to put the industrial disease prevention program across.

Fortunately or unfortunately for that conscientious lady, there is so much truth in the statement that she fails to realize that we all are "passing the buck" to her.

The Industrial Nurse

—Her Responsibility in the Efficient Administration of a Compensation Law—

B. E. KUECHLE,
Vice-President and Claim Manager,
Employers Mutual Liability Insurance Company,
Wausau, Wisconsin

IN A NARROW sense, the administration of compensation laws has been placed by the legislatures of various states under the jurisdiction of Industrial Commissions, Industrial Accident Boards, or Labor Departments. In a still narrower sense, the administration of such laws merely implies a supervisory and a quasi-judicial authority to bring compensation and medical benefits to employees injured in industry or to their dependents.

However, in the broad sense of the term, the efficient administration of a compensation law relegates the compensation benefit idea more or less to the background as a penalty imposed on industry because of a breakdown somewhere along the line which made possible the accidents or diseases for which compensation becomes payable.

This idea was forcibly expressed by the committee which drafted the Wisconsin Workmen's Compensation Law, the first law of its kind in the country. This committee listed as one of the objects to be attained in the passage of a workmen's compensation law, the reduction of accidents and injuries in industrial pursuits. To obtain this objective requires a comprehensive understanding of the entire problem of not only industrial health, which includes industrial accidents and diseases, but public health. The responsibility rests not alone with industrial boards, but also with industry, labor, organized medicine, hospital associations, nursing associations, public and private health agencies. Even more than that, it requires the cooperation of the public and each individual member of the public.

MY assignment is to select from this large group the industrial nurse and point out where she fits in the picture, and what her responsibility is in efficiently administering compensation laws. A few figures I have read within the past few weeks indicate that there is still a big job to be done, and the surface has only been scratched.

The Defense Commission recently released some information showing that we still have annually in this country 17,000 industrial deaths, 75,000 major permanent disabilities, and 1,400,000 lesser injuries.[1] It has been estimated that benefits paid under compensation laws of this country annually are approximately $400,000,000.

The National Council on Compensation Insurance shows incurred compensation costs for all insured business in 1936 as $208,420,062.[2] This does not include self-insured losses or payments by several state funds. Therefore, it has been estimated that the inclusion of all incurred losses for 1940 would practically double the insurance payments for 1936.

Actuaries have estimated that compensation benefits represent only about 25% of the economic loss occasioned by industrial accidents and diseases.[3] Concurrent economic losses are represented by increased labor turnover necessitated by accidents, damaged machinery, damaged materials and products, and decreased efficiency of plant operation.

In spite of these staggering figures, it has been estimated that the economic loss caused by non-industrial diseases exceeds by ten-fold the loss caused by both industrial accidents and diseases. DR. C. O. SAPPINGTON, Chicago industrial hygienist, in addressing the recent meeting of the American Conference of Industrial Physicians in Chicago stated that "40,000,000 workers in the United States

Delivered at a Symposium on Industrial Public Health Nursing Services, Hotel Wisconsin, Milwaukee, Wisconsin, February 20, 1941.

1. U. S. Public Health Service, Washington, D. C., November 10, 1940. Printed in *Public Health Nursing*, December, 1940.
2. National Pure Premiums, Revision No. 8.
3. H. W. HEINRICH, Page 8, 4th Biennial Bulletin, Minnesota Compensation Insurance Board.

lose 280,000,000 working days each year because of ill health. It has been estimated that the average loss of time per employee per year due to sickness is seven days. At $28 per person per year, this amounts to an aggregate of $1,120,000,000 for 40 million workers. This is merely the loss due to unpaid wages on account of sickness among industrial employees, and does not include disbursements for medical and nursing care, hospital bills, labor turnover, idle machines, and other items which go to make up a total of 10 billion dollars lost because of ill health among industrial workers each year."

Truly, this is a challenge which merits our best efforts, especially in these times of national stress and emergency.

FOR a moment now, let's look at the more encouraging side of the picture. I think it is quite generally conceded that Wisconsin is just about "tops" nationally, not only in its accomplishments in industrial accident and disease prevention, but in its accomplishments in the industrial and public health field. Factual information is available in support of this statement. Within the past month the Wisconsin State Medical Society has released some figures illustrated by the chart you see before you which I have taken the liberty of reproducing. It is truly amazing to think that compared with the nation-wide absence of workers due to illness, Wisconsin has this unbelievable record of only 20% as much absence as all of the other states in the union combined. A few of these charts graphically show the outstanding achievements in our state in the field of industrial accidents. In a great measure, this unusual record has been due to the fine leadership of the Wisconsin Industrial Commission and the splendid cooperation of industry, labor, organized medicine, and the insurance carriers. The record has made it possible for the Wisconsin legislature to increase compensation insurance benefits by practically 100% since the law was passed in 1911, with only a 27% increase in insurance rates as compared with 1911. By comparison, look at this chart of comparative compensation law levels and insurance costs in Wisconsin and a few other industrial states.

In spite of the substantial increase in employment in Wisconsin in 1940 because of the defense program, last year Wisconsin had only 113 industrial deaths. With the exception of 1934 when employment was low because of the depression, this is the smallest number of industrial deaths ever recorded in this state. On the basis of the figures previously referred to, that we have in this nation about 17,000 industrial deaths per year, Wisconsin should, on the basis of the number of the employees in industry, reasonably expect to have about 400 such deaths.

This all goes to show that while the cost problem is still almost beyond human comprehension, a concerted effort such as has been practiced in Wisconsin for many years does produce worthwhile results.

There can be no doubt but that the industrial nurse has been a big factor in making this record possible in Wisconsin. I have been told that at the present time there are 165 industrial nurses employed in 140 establishments. I well remember the day when our company first induced one of our large policyholders to employ an industrial nurse. Not only was it a difficult job to sell the employer, but we met with considerable opposition from the physicians servicing that plant. The big difficulty in selling such a program to industry was because in their past experience, they had been led to believe that the capacities of the usual nurse were limited to those duties which are carried out in connection with patients. I know of no instance where the services of an industrial nurse have been dispensed with, even under the financial stress of the depression, when the employer was properly sold on the endless activities in which the nurse might engage. The nursing profession is in need of a campaign by which the employers may be educated to realize the extent to which the executives of the small plant may be relieved of many personnel problems. The field of the industrial nurse is almost unlimited.

A practical industrial nurse will concern herself with such problems as fatigue, noise, vibrations, posture requirements at work, odors, the influence of extreme heat or cold, air conditioning, protective garments, eating facilities, uniforms for workers, rest periods, dust, gases, smoke, fumes, hours of labor, absenteeism, physical types in relation to work requirements, variations in susceptibility to disease of different types of workers, differences in capacities of men and women, industrial neurosis, alcoholism, and home conditions in relation to work. In addition to their generally accepted jobs relating to patients, most good industrial nurses are now taking an active interest in most of the items that I have just mentioned.

NATURALLY, no nurse should carry out her specific nursing duties except under the direction of the plant physician, whether he is on full time, part time, or only connected with the plant in a consulting capacity. She should be careful not to overstep the bounds of medical ethics and to avoid doing so, should, if possible, work under written or printed standing orders approved by the physician. If the nurse's activities are confined solely to this field, however, her contact with employees will be rather limited and may not justify her employment. It has been estimated that under such circumstances, probably not in excess of 2% or 3% of the employees will make use of the nursing facilities each day. However, where the nurse accepts her broader responsibilities, the number of workers passing through the plant hospital will increase up to 400%.

No one has a better opportunity than the industrial nurse to learn the real story of how accidents actually happen. No accident prevention program can be effective unless the facts behind the acci-

dent become known. Therefore, the nurse should be familiar with operating conditions in the plant so that she may intelligently extract from the employee the real cause of the accident. She should be a member of the plant's safety committee in order that the information she has received from employees may be capitalized upon to prevent the occurrence of similar accidents. Complaints of illness by employees should be interpreted into possible hazardous exposures in the plant, which, when corrected, may avoid similar illness among other employees.

NO health or safety program can be complete unless securely anchored on a comprehensive physical examination program. This is true even though the particular plant does not have an occupational disease hazard. The industrial nurse can exert more influence on the management of her organization to adopt such a program than anyone else.

Since any physical examination program, in order to be successful, must safeguard the rights of workers, the nurse because of her close contact with employees, must be the sales agency through which labor will wholeheartedly support such a program when adopted.

Every nurse should be reasonably familiar with the principal provisions of her state compensation law and should have a copy of that law readily available. Employees constantly seek advice about their rights under such a law and the nurse should be able to answer such inquiries intelligently.

Naturally, complete records should not only be made of all employee visits, but they should be of a permanent nature so they will be available on any future occasion.

It is usually the duty of the nurse to make reports of accidents to insurance carriers and industrial boards. Reports should be made out promptly, intelligently, and completely so that compensation payments may go forward promptly when money is most needed.

The nurse should familiarize herself with plant operations so that employees may be encouraged to rehabilitate themselves more promptly and effectively by part-time work as soon as their physical condition warrants part-time employment.

Confidence of a patient in his physician is a big factor in prompt recovery. Many employees have had little contact with physicians prior to an industrial accident. The assistance of the nurse in such a case, not only in the selection of a qualified physician, but also in building confidence in the physician chosen is of special importance. This interest in the patient should continue throughout his disability.

Above all else, let's remember that no comprehensive program as outlined can be successful unless management is thoroughly sold, not only on the financial benefits to be derived, but on the humanitarian motive as well.

No one is in better position to bring this story home to management than a psychologically equipped industrial nurse.

Industrial Poisonings

—*Practical Aspects*—

MAX TRUMPER, PH.D.,
Chief of the Department of Biochemistry and Toxicology, St. Luke's and Children's Medical Center,
Philadelphia, Pennsylvania

IT IS my purpose to present cases of poisoning from various industries to show the need for correlating with the clinical findings a thorough occupational history which should include the working conditions of the plant. This correlation presupposes a general knowledge of the chemical and physical processes of a given industry.

Two hundred and forty years ago physicians were urged by Bernardini Ramazzini to ask their patients, "What is your occupation?" His pioneer book in Latin on Diseases of Workers has recently been published with an inter-leaf translation. Since then, owing to the industrial revolution, this question as to what is your occupation is not so simple as it was in the time of Ramazzini. For in recent years the spectacular advances in creating new organic compounds and in the manufacture of the synthetic chemicals have brought with them an increase in the complexity of the toxic materials used in industry, along with the introduction of new solvents of uncertain or unknown toxicity. All of this presents a problem that needs the team-work of physicians, chemists, and engineers. This is especially so in communities characterized by diversified industries.

Today the doctor must ask the industrial worker many detailed questions as to the nature of his work. We cannot rest content with noting that the patient is a carpenter and therefore should be free from toxic exposures. Frequently it is essential to seek out more intelligent workers or foremen who can supply the necessary information, otherwise unknown or obscure intoxications will go unrecognized. If, for one reason or another, the physician cannot go into the many details of the patient's occupation, it is desirable to obtain the necessary information from some one who has inspected the plant or has a technical knowledge of the processes and materials used in that industry. I will try to illustrate the need for this correlation by summarizing specific instances of poisoning which I have encountered in several industries.

DURING the past six years I have been interested in carbon disulfide poisoning. It is surprising the wide range in degrees of safety found in the different viscose-rayon plants in this country. Fortunately during the past few years in Pennsylvania there have been more safety and protective devices installed than in the previous 20 years.

Perhaps I can illustrate my point best by quoting from Dr. Alice Hamilton: "A few years ago I

This paper was presented February 3, 1941, before the Section on Public Health, Preventive and Industrial Medicine of the College of Physicians, Philadelphia.

had a telegram sent to me at Harvard, not from a physician but from a registered nurse saying, 'Epidemic of insanity in artificial-silk mill. Doctors do not understand. Can you tell us anything?' I wrote back voluminous information. Then I tried to find out what had happened, but it was shrouded in secrecy. I have never heard. There is no sequel to that story at all."

The sequel is as follows: We had examined three of the above-mentioned cases in February, 1934, and another three in 1935. These patients worked in a plant which had been changed over into a viscose plant; some 40 women employed in the reeling room developed acute symptoms, with an epidemic of hysteria. The symptoms were excitement bordering on hysteria, yelling, fainting, and acute psychoses. It soon became obvious that something was seriously wrong. One of the chemists who analyzed the air of the reeling room used to get sick while collecting his samples of air. The ventilating efficiency of that room was then stepped up 25% above that of any other viscose plant. Nevertheless at intervals there were toxic amounts of carbon disulfide in that reeling room, as confirmed by the symptoms of some of the workers. Both the forelady and the foreman in the room were poisoned. We discovered that the concentrated carbon disulfide vapors were regularly exhausted from the churn room at a point less than 100 feet from the large and powerful fresh air intake of the reeling room. The exhaust from the churn room was about 30 feet above the ground level and the vapors, being heavy, would tend to drop. It happened that the prevailing winds made certain that at times sufficient carbon disulfide would be carried in front of the reeling room intake and become part of the fresh air in the reeling room, to give rise to the acute symptoms. This situation has since been remedied.

LET us now consider carbon tetrachloride and naphtha which are widely used solvents. In making an inspection of a metropolitan newspaper plant where the primary problem, as you all know, is that of lead exposure, I recognized the odor of carbon tetrachloride. I found it was being used as a degreasing agent in the mechanical repair department where about six men were employed. Nothing unusual was noticed the first day, but on the subsequent days I noticed the odor persisted and was more marked after the degreasing process.

This firm had used naphtha as the degreasing agent for a great many years, but one of the representatives from the fire underwriters insisted that the naphtha be mixed with an equal amount of carbon tetrachloride to reduce the hazard from fire. The ventilation in this machine shop was a 24-inch fan located near the ceiling. This was satisfactory when naphtha was used. But when the heavy tetrachloride was added (five times heavier than air) the exhaust from the fan retarded the rate of tetrachloride sedimentation. Some of the diluted vapors were drawn up to the breathing level of the mechanics. Nearly all of the workers when questioned admitted digestive complaints, especially nausea, and a few vomited and complained of being dizzy. One of these workers was definitely affected and presented a train of symptoms characteristic of the poison. He was the only man who showed neurological symptoms and was placed under the care of a neurologist, experienced with lipoid solvents. He was the best mechanic in the department. It is not unusual from the toxicological viewpoint to find that the hardest worker is often the first to show poisoning. This may happen because he is more apt to be a trouble shooter or perhaps the hardest worker. As you know the average adult breathes:

Minute Ventilation of a Man 68 kilos (150 pounds)	
Rest in bed, fasting	6 liters per minute
Sitting	7 liters per minute
Standing	8 liters per minute
Walking two miles an hour	14 liters per minute
Walking four miles an hour	26 liters per minute
Maximum exertion	65–100 liters per minute

To remedy this situation we placed the degreasing operation in a booth outside the machine shop. A fan was installed at the work bench one-third above the work level and two-thirds below. This eliminated any trace of carbon tetrachloride and was followed by a disappearance of the complaints of nausea and vomiting, as well as the dizziness and eye complaints.

I now wish to refer to another instance where carbon tetrachloride was the wrong solvent to use because it is readily trapped whenever down draft ventilation is not available. There were approximately 100 carpenters and joiners engaged in tailoring and cementing slabs of insulating cork against the metal walls of ship bulkheads, magazines and refrigerator rooms. Each man used about five gallons of this cement a day, smearing and spreading it with a trowel to a thickness of about one-eighth of an inch. The cork was one-inch thick, 12 inches wide and 36 inches long, and the cement had a consistency like cold molasses, and contained 24% carbon tetrachloride. Much carpentering work was required to shore up these blocks of cork until the cement dried. At first no respirators were given the men, but later as the complaints increased the men were given respirators when working with the cement. Unfortunately, a few days later when they returned to remove the shoring no respirators were worn. Thus further exposure followed because these heavy vapors were trapped in the various quarters 20 to 40 feet below deck where the blowers stirred up and diluted the vapors but did not vent the accumulated vapors.

I obtained an occupational history from 13 of these men when it became obvious that they were all showing varying degrees of CCl_4 intoxication, two of these men had been acutely ill, one of whom had an exploratory laparotomy. Their complaints were: "nausea and vomiting, stomach could not hold anything, lungs were always sore, headaches and dizziness, sore eyes and throat, troubled dreams at night"; one emphasized it made him at first voraciously hungry — "just couldn't get enough to eat but by the end of the

week couldn't keep my lunch down." A few were jaundiced. One fellow used the words "everything went black — passed out — after I worked with it only two and a half hours."

When this situation was brought to the attention of the employer 90% of the carbon tetrachloride was replaced with naphtha, the latter being lighter was much easier to ventilate. There were no further complaints so far as I know.

These men were all carpenters or joiners. Ordinarily we think of their work as being practically free from serious toxic hazards and only an occasional exposure to small quantities of solvents.

I NOW wish to refer to two instances in which the exposures to lead were unusual. In some instances even though your patient does give you intelligent answers to your questions as to the many details of his occupation it is desirable to make an inspection of the plant before arriving at your conclusion. Perhaps I can best illustrate this correlation by the following situation: A wholesale glass firm employed from 75 to 100 men, five to 10 of whom were engaged in the manufacture of mirrors. The men in the mirror department had a high incidence of illness and the quality of their work was likewise unsatisfactory. It looked like a hopeless task, after I made a thorough inspection of this small plant. As part of the routine I examined the drinking water, which was satisfactory. I also tested the distilled water, because pure water is essential in making good mirrors. I was surprised to find the distilled water rich in lead and iron.

Here was a situation that could easily have developed into a serious outbreak of lead poisoning, for many of the employees had been drinking this so-called pure water.

We found that an unscrupulous plumber had been called in to repair a leak in the large tin condenser which was housed in a hogshead barrel. Unknown to the firm he had replaced this costly condenser made from high grade lead-free tin with soft inexpensive lead piping. Thus the hot steam from the boiler on reaching the coils of lead pipe housed in the hogshead barrel dissolved considerable amounts of lead. Fortunately, the presence of the lead in the distilled water also interfered with the quality of the mirrors that were being manufactured and so this situation was corrected before any serious poisoning resulted.

This is the case of a colored man, aged 46, whose medical history was entirely negative except that he had gonorrhea in 1912 and also had anti-luetic therapy consisting of 32 injections. The patient came into the accident ward of the St. Luke's and Children's Hospital September 13, 1939, in a fixed crouched position, after vomiting twice and he complained of severe generalized abdominal pain. A few days later the entire abdomen was rigid, more marked in the right upper abdomen. One week later the abdomen was still rigid with extreme tenderness over the gall-bladder area. The only day the patient was free from abdominal pain was when he received 10 cc. intravenously of 10% calcium gluconate. In view of the fixed pupils and the absence of knee jerks, tabetic crisis was suspected and it was deemed advisable not to operate. He was discharged September 30, 1939. One year later he was re-admitted with costovertebral pain, on the right side which radiated down to the lower right quadrant. A tentative diagnosis of renal colic, possibly lead colic, was made. He felt as if something fell from the kidney and complained of severe pain in the lumbar region posteriorally. He was given 10 cc. of calcium gluconate and then received intravenously 5% glucose in saline. During this medication his pain again receded. At the operation, performed by Dr. H. L. Weinstock, a small stone was found just below the right uretero-pelvic junction. Since the x-ray revealed some calcification or possible stone in the left lower pelvis, Dr. Weinstock prepared for a second operation, but the patient refused operation despite a non-functioning left kidney.

Now let us examine the answers to the question: What is your occupation? He was employed as a laborer in the corroding white lead department of a plant which still used the Old Dutch process (one which gives a maximum exposure to lead). This department of the plant was noted for its high lead dust exposure, as confirmed by the extreme dustiness of the work and the heavy accumulation of lead dust on walls, pipes, etc.

There was also a high incidence of from moderate to severe lead poisoning among his fellow workers. In this regard it is interesting to note that on October 5, 1939, when the urine was first analyzed for lead, only 0.054 milligrams per liter of urine was found, which was within the so-called normal range. Since there was some rise in blood urea and creatinine, the normal urinary lead findings were not considered significant.

It seemed to me that here was an individual with a moderately severe exposure to lead dust from whom a typical phosphatic calculus weighing 190 milligrams had been removed. Knowing how closely calcium and lead metabolism parallel each other it occurred to me that this calculus might contain some lead. Analysis showed it did contain 3% of lead. There is one additional feature of this case, that is the unusual etiology. The question is whether the lead found in the renal calculus was occupational or non-occupational in origin, or possibly both. As revealed by the roentgenological report of Dr. Joseph W. Post, this patient still carries "bird-shot" in his right forearm, left leg and thigh. His occupational exposure to lead dust was five and a half years while his "bird-shot" exposure was for a period of 23 years. After the remaining calculus has been removed we may be able to answer this question from a study of the impurities present in the metal, since antimony is used to harden lead pellets employed as bird or buck shot. A more detailed report on this case by Dr. S. T. Gordy and myself will appear in *J.A.M.A.* As far as we have been able to learn from a careful search of the American and foreign literature, there has been no previous report of a urinary calculus which contained lead.

IN CONCLUSION, I have tried to show from a few instances in my experience the need for correlating with your clinical findings a thorough occupational history which should include wherever possible an inspection of the working conditions of a given industry, particularly in communities with diversified industries, because we are in for a period of maximum productivity and increased hours of work.

The Pittsburgh Meeting

—*Institute of Industrial Medicine and Industrial Hygiene*—

FOLLOWING is the complete program of the annual meetings to be held in Pittsburgh, Pennsylvania, May 5, 6, 7, 8, and 9, 1941 — the Twenty-Sixth Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, and the Second Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION:

Monday, May 5

REGISTRATION—No Fee
9:00–10:00—WILLIAM PENN HOTEL
LECTURES AND DEMONSTRATIONS
MERCY HOSPITAL, Stevenson and Locust Street, Pittsburgh
JOHN P. GRIFFITH, M.D., *Chairman*
10:00–12:30—Mercy Hospital Staff
12:30– 1:30—Lunch
1:30– 4:30—Mercy Hospital Staff
Monday will be devoted to a series of Dry Clinics in Industrial Medicine and Surgery at Mercy Hospital.

Tuesday Morning, May 6

9:00—ADDRESS OF WELCOME
HON. CORNELIUS D. SCULLY, *Mayor, City of Pittsburgh*
9:15—ADDRESS OF WELCOME
FREDERICK JACOBS, M.D., *President, Allegheny County Medical Society*
9:25—ADDRESS OF WELCOME
JOHN T. SHAW, M.D., *Secretary of Health, Commonwealth of Pennsylvania*
9:35—RESPONSE
DANIEL L. LYNCH, M.D., *President of the American Association of Industrial Physicians and Surgeons*
9:40—RESPONSE
MR. WARREN A. COOK, *President of the American Industrial Hygiene Association*

ANNUAL MEETING—AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

9:45–10:10 President's Address, DANIEL L. LYNCH, M.D.
10:10–10:30 "Some Practical Results of Periodic Physical Examinations"—A. J. KAMMER, M.D., *Medical Director, Inland Steel Company*, Indiana Harbor, Indiana
10:30–10:40 Discussion
10:40–11:00 "Relation of Trauma to Disease"—A. H. COLWELL, M.D., *Associate Professor of Medicine, School of Medicine, University of Pittsburgh*
11:00–11:10 Discussion
11:10–11:40 "Contact Dermatoses"—W. H. GUY, M.D., *Professor of Dermatology, School of Medicine, University of Pittsburgh*
11:40–12:00 Discussion
12:30– 2:00 Luncheon and Exhibits

Tuesday Afternoon, May 6

2:00– 2:20 "Sequelæ of Head Injuries"—S. S. ALLEN, M.D., Pittsburgh
2:20– 2:30 Discussion
2:30– 2:50 "Trauma Other Than Fractures"—J. H. WAGNER, M.D., *Carnegie-Illinois Steel Corporation*, Pittsburgh
2:50– 3:00 Discussion
3:00– 3:20 "Mental Hygiene in Industry"—C. H. HENNINGER, M.D., *Professor of Psychiatry, School of Medicine, University of Pittsburgh*
3:20– 3:30 Discussion
3:30– 3:50 "Evaluation of Circulatory Function in Extremities"—M. B. FERDERBER, M.D., *Department of Industrial Hygiene, School of Medicine, University of Pittsburgh*
3:50– 4:00 Discussion
4:00– 4:30 Business Session
4:30– 6:00 Exhibits

Tuesday Evening, May 6

INDIVIDUAL GROUP CONFERENCES

Wednesday Morning, May 7

9:00– 9:20 "Medical and Preventive Aspects of Carbon Monoxide Poisoning"—F. S. ROSSITER, M.D., *Carnegie-Illinois Steel Corporation*, Pittsburgh
9:20– 9:30 Discussion
9:30–10:00 "Recent Investigations of Effects of Radiation from an Ophthalmologic Viewpoint"—C. F. KUTSCHER, M.D., Pittsburgh
10:05–10:35 "The Absorption and Excretion of Ingested Lead Under Various Experimental Conditions"—R. A. KEHOE, M.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati*, Cincinnati, Ohio
10:45–11:05 "Engineering Control of Lead Contaminated Environments"—G. C. HARROLD, Ph.D., *Industrial Hygiene Laboratories, Chrysler Corporation*, Detroit, Michigan
11:10–11:25 "Lead Exposures in the Printing Industry"—R. T. HOMEWOOD, *Virginia Department of Health*, Richmond, Va.
11:30–11:45 "Incidence of Lead Poisoning in the City of Baltimore"—E. KAPLAND, Ph.D. and J. M. McDONALD, M.D., *Baltimore Health Department*, Baltimore, Md.
11:45–12:00 Discussion
12:00– 2:00 Luncheon and Exhibits

Wednesday Afternoon, May 7

2:00– 2.20 "Tuberculosis and Its Allied Conditions as Commonly Seen in Our Industrial Population"—C. H. MARCY, M.D., *Director Tuberculosis League Hospital*, Pittsburgh
2:20– 2:40 "Relationship between Tuberculosis and Inhalation of Various Substances from a Pathological Standpoint, Including Bituminous Coal Dust Inhalation"—S. R. HAYTHORN, M.D., *Director Singer Laboratory, Allegheny General Hospital*, Pittsburgh
2:40– 2:50 Discussion, R. R. SAYERS, M.D., *Director U. S. Bureau of Mines*, Washington, D. C.
2:55– 3:20 "Engineering Preventive Methods in the Control of Tuberculosis"—D. E. CUMMINGS, *Director Division of Industrial Hygiene, University of Colorado*, Denver, Colorado
3:25– 3:45 "Control of Tuberculosis and the Employment of a Tuberculous Individual in Industry"—W. A. SAWYER, M.D., *Medical Director, Eastman Kodak Company*, Rochester, N. Y.
3:45– 3:55 Discussion
3:55– 4:20 "Bacteria in Air and Disinfection"—A. E. WILLIAMSON and H. B. GOTAAS, *Department of Sanitary Engineering, School of Public Health, University of North Carolina*, Chapel Hill, N. C.
4:20– 4:35 "Variation in Bacterial Population in Certain Ventilating Systems"—A. HOLLAENDER, Ph.D., *National Institute of Health*, Bethesda, Md.
4:35– 4:45 Discussion
4:45– 6:00 Exhibits

Wednesday Evening, May 7

6:30—BANQUET

"The Role of Industrial Medicine and Industrial Hygiene in National Defense"—IRVIN ABELL, M.D., *Chairman of Health and Medicine Committee, Federal Security Agency*, Washington, D. C.

"Noise and Hearing"—V. O. KNUDSEN, Ph.D., *Dean of the Graduate School, University of California*, Berkeley, California

WESTINGHOUSE QUARTET

Thursday Morning, May 8

8:30– 8:50 "Design of Lateral Exhaust Hoods for Industrial Gases, Vapors and Fumes"—L. SILVERMAN, *Harvard School of Public Health*, Boston, Mass.

8:55– 9:15 "Ventilating for Electroplating Operations"—A. D. BRANDT, Sc.D,. *National Institute of Health, Division of Industrial Hygiene and Sanitation*, Bethesda, Md.

9:15– 9:25 Discussion

9:25– 9:50 "Airborne Infection and Disinfection in Air Conditioning"—W. F. WELLS, *University of Pennsylvania*, Philadelphia, Pa.

9:55–10:20 "Local Cooling of Workers in Hot Atmospheres"—F. C. HOUGHTEN and M. B. FERDERBER, M.D., *American Society Heating and Ventilating sylvania*, Philadelphia

10:25–10:50 "Radiant Cooling"—C. P. YAGLOU and O. SCHNEIDER, PH.D., *Harvard School of Public Health*, Boston, Mass.

10:50–11:05 Discussion

11:05–11:20 "Comparison of Quick Methods for Quantitation of Impinger Samples of Granite Dust"—C. L. POOLE, *Rhode Island State Department of Health*, Providence, R. I.

11:25–11:40 "An Immersion Method for the Rapid Approximation of Quartz in Granite"—W. B. WARDLOW, *Texas State Board of Health*, Austin, Tex.

11:45–12:00 "Use of X-ray Diffraction Equipment to Determine Silica in Dust Exposures"—J. W. BALLARD, Ph.D., *U. S. Bureau of Mines, Pittsburgh Experiment Station*, Pittsburgh

12:00–12:15 General Discussion

12:15– 1:30 Luncheon and Exhibits

Thursday Afternoon, May 8

1:30– 1:45 "The Effects of the Inhalation of Dusts Encountered in the Chrome Brick Industry"—W. B. FULTON, M.D., and F. E. BUTTERS, *Division of Industrial Hygiene, Pennsylvania Department of Health*, Harrisburg, Pa.

1:50– 2:05 "Engineering Survey in Tri-State Lead and Zinc Mining District"—E. C. WARKENTIN, *Oklahoma State Department of Health, Division of Industrial Hygiene*, Oklahoma City, Okla.

2:10– 2:25 "Industrial Hygiene Control on Tunnel Construction in Hydro-Electric Projects in Western North Carolina"—M. F. TRICE, *Industrial Hygiene Engineer, Division of Industrial Hygiene, North Carolina State Board of Health*, Raleigh

2:25– 2:40 Discussion

2:40– 2:55 "Etiological Aspects of Particle Size in Quartz Dust" — B. D. TEBBENS, and R. SCHULTZ, M.D., *Harvard School of Public Health*, Boston, Mass.

3:00– 3:20 "Particle Sizes of Dust as Found in Various Industries and Determination of Silica in Bituminous Coal Dusts"—C. E. BROWN, Sc.D., and H. H. SCHRENK, PH.D., *U. S. Bureau of Mines, Pittsburgh Experiment Station*, Pittsburgh

3:20– 3:30 Discussion

3:30– 3:45 "Animal and Petrographic Studies on Talc Dusts"—C. WILLIAMS, PH.D., *Liberty Mutual Ins. Co.*, and R. SCHULTZ, M.D., *Harvard School of Public Health*, Boston, Mass.

3:50– 4:05 "Fuller's Earth as a Cause of Pneumoconiosis"—W. D. MCNALLY, M.D., *Consulting Toxicologist*, Chicago

4:10– 4:35 "Silicosis Among Naval Foundry Men"—E. W. BROWN, M.D., and W. E. KLEIN, M.D., *Medical Corps, U. S Navy*

4:35– 5:00 Discussion

5:00– 6:00 Exhibits

Thursday Evening, May 8

7:30– 8:00 "Review of Industrial Hygiene Progress During 1940"—WARREN A. COOK, *President, American Industrial Hygiene Association*

8:00– 8:15 "Claims of Industrial Poisoning"—P. N. HARGER, Ph.D., *Prof. of Biochemistry and Toxicology, University of Indiana, School of Medicine*, Indianapolis, Ind.

8:20– 8:35 "Carbon Disulfide and Hydrogen Sulfide Exposure in the Synthetic Sausage and Casing Industry using the Viscose Process"—MILTON H. KRONENBERG, M.D., KENNETH MORSE, M.S., ALFRED SETTERLIND, M.S., *Division of Industrial Hygiene, Illinois Department of Health*, Chicago

8:40– 8:55 "Sampling and Determination of Aldehydes"—E. C. BARNES, *Industrial Hygiene Laboratory, Westinghouse Electric and Manufacturing Company*, East Pittsburgh, Pa.

9:00– 9:25 "The Absorption and Elimination of Noxious Vapors"—H. W. HAGGARD, M.D., *Director of Laboratory of Applied Physiology, Yale University*, New Haven, Conn.

9:25 Discussion

Friday Morning, May 9

8:30– 8:45 "Dermatitis Control in a Mass Production Industry"—T. F. MOONEY, *Ford Motor Co.*, Dearborn, Mich.

8:50– 9:05 "Sporotrichosis as an Occupational Disease Among Florists"—L. W. SPOLYAR, M.D., *Indiana State Board of Health, Industrial Hygiene Division*, Indianapolis, Ind.

9:10– 9:35 "A Statistical Approach in the Study of Hematologic Data from Some Common Solvent Exposures"—J. H. STERNER, M.D., *Eastman Kodak Co.*, Rochester, N. Y.

9:40– 9:55 "Recovery Following Exposure to Benzol"—L. J. GOLDWATER, M.D., *New York University, Dept. of Preventive Medicine*, New York City.

9:55–10:10 Discussion

10:10–10:30 "Electric Arc Welding, Coated Rods"—C. P. MCCORD, M.D., S. F. MEEK, M.D., and G. C. HARROLD, Ph.D., *Industrial Hygiene Laboratories, Chrysler Corporation*, Detroit, Mich.

10:35–10:55 "Gases, Fumes and Vapors from Coated Welding Rods"—B. D. TEBBENS, Sc.D., and PHILIP DRINKER, *Harvard School of Public Health*, Boston.

10:55–11:05 Discussion

11:05–11:25 "Trends in Occupational Disease and Industrial Hygiene"—H. B. MELLER, Sc.D., and F. S. MALLETTE, *Air Hygiene Foundation, Mellon Institute*, Pittsburgh

11:30–11:50 "The Human Eye and Its Protection in Industry"—W. H. LEHMBERG, Sc.D., *American Optical Company*, Southbridge, Mass.

11:50–12:00 Discussion

12:00– 1:30 Exhibits

Friday Afternoon, May 9

ANNUAL MEETING, SECOND SESSION

1:30– 1:50 Election of Officers

1:55– 2:10 "Controlled Radium Hazards" — FREDERICK FLINN, Ph.D., *Columbia University*, New York City

2:15– 2:35 "Some Recent Observations on Phosphorus and on Ammonium Nitrate Toxicology"—R. B. L. FLEMING, *Tennessee Valley Authority*

2:40– 2:55 "Biochemistry of Carbon Bisulfide"—R. W. MCKEY, PH.D., *Harvard School of Public Health*, Boston, Mass.

2:55– 3:10 Discussion

3:15– 3:30 "Toxicity of 1-1 Dichlor-1-nitroethane"—WILLARD MACHLE, M.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati*, Cincinnati, Ohio

3:35– 3:50 "Acute Toxic Effects Upon Experimental Animals of Methyl, Ethyl and Butyl Methacrylates"—WILHELM DEICHMANN, Ph.D., *Kettering Laboratory of Applied Physiology, University of Cincinnati*, Cincinnati, Ohio

3:55– 4:10 "Toxicity of Methyl Silicate"—W. G. FREDRICK, Ph.D., *Industrial Hygiene Bureau*, Detroit.

4:15– 4:30 Discussion

4:30– Business Meeting (Annual)

American Association
of Industrial Physicians and Surgeons

—Constitution and By-Law Amendments Proposed to be Voted upon at the Annual Meeting, May 5-9, 1941—

CONSTITUTION:

ARTICLE V, SECTION 3: After the words "twelve directors" in line 4, insert "and a House of Delegates consisting of the Officers and members of the Board of Directors and the Presidents or legally appointed or elected delegates of Component Societies." Delete that part of Section 3 beginning with "Presidents of Component Societies" and ending with "as other directors."

ARTICLE V: Change heading to read "Officers, Directors and Governing Bodies."

ARTICLE V, SECTION 1: After the words "Second Vice-President" insert "a Managing Director" and delete the word "Secretary" in the compound word "Secretary-Treasurer."

ARTICLE V, SECTION 4: Delete entire section which will be placed in the By-Laws.

ARTICLE V, SECTION 5 becomes Section 4, and change the word "five" to "six."

BY- LAWS:

ARTICLE I, SECTION 2: Change it to read "Special meeting of the membership may be called by the President with the approval of five members of the Board of Directors. Special meetings of the Board of Directors or of the House of Delegates may be called at the discretion of the President."

ARTICLE I, SECTION 3: Insert after the words "all meetings" the words "of the members" and delete the word "Treasurer" in the compound word "Secretary-Treasurer."

ARTICLE I, SECTION 4: Delete the word "Treasurer" in the compound word "Secretary-Treasurer."

ARTICLE IV: Change heading to "Duties of Officers and Governing Bodies."

ARTICLE IV, SECTION 1: "The planning of the work of the Association; arrangements for meetings and programs and any or all other matters pertaining to the administration of the affairs of the Association shall originate in the House of Delegates but all its actions must be ratified by the Board of Directors."

ARTICLE IV, SECTION 2: "The Board of Directors shall have charge of the property and financial affairs of the Association and shall perform such duties as are prescribed by the Constitution and By-Laws and the State law governing directors of corporations. It must ratify the transactions of the House of Delegates, Committees and Boards before they can become effective."

All sections to be renumbered: Section 1 becoming Section 3, etc.

ARTICLE IV, SECTION 4: "The Managing Director shall be the executive officer of the Executive Committee and, under its supervision, during the intervals between meetings, shall manage, direct and conduct the affairs of the Association. He shall supervise and direct the activities of the Executive Secretary and shall render a report on the status of the Association to the Board of Directors at their Annual Meeting."

ARTICLE IV, SECTION 2 and 3: Delete the word "Secretary" in the compound word "Secretary-Treasurer" and the last one in section 3 should be changed to "Managing Director."

Presenting the Case for a Health and Medical Plan

HAROLD A. VONACHEN, B.S., M.D.,
Medical Director Caterpillar Tractor Company, Peoria, Illinois

REGARDLESS of the many articles and essays on industrial health, we must still assume that industrial medicine is in its infancy, awaiting adoption and growth by men who are far sighted enough to realize that medical plans of their own making are far more efficient than those fostered and born of legislation. Laws result from neglect in providing our own programs.

Large industries have made remarkable strides in industrial medicine, but for every so-called large industrial plant there are a hundred smaller industries employing many more individuals who are entitled to the same safeguards of health and who are merely waiting for some group to lead the way and "sell" the idea of a well organized plan that will function without enormous expense. In the near future it will be necessary for the men who have been trained in industrial health organization to give freely of their time in order that others may profit by their experience. Incidently, a recent survey revealed that in the United States today there are less than 200 physicians who are capable of teaching and instructing the smaller industries in this important work.

That industry is ready and willing to cooperate if shown the way, is revealed by a recent statement of B. C. HEACOCK: "Actually, if a firm can afford to stay in business, it can afford a medical program, whether it employs six or six thousand. We would have to look a long way before we could find an investment that is sounder than an investment in health."

FRANKLY, this article is written for but one purpose, that being to present in as few words as possible certain facts and plans to industries that are yet waiting to start or to improve their health and medical programs. The facts and figures given art the result of 14 years of experience in building a program which is not perfect, nor will it ever be, but one organized from our own experiences and those of many others.

It is true that an industrial health program can be justified from a standpoint of dollars and cents, but most employers seek benefits primarily of a human, rather than a monetary nature. The purpose of a health program, after all, is not to make money but to make health. Any financial advantage is essentially a by-product and must always be recognized as such.

Now let us consider the minimum aspects and requirements for an industrial health program, beginning with the individual as he

presents himself for employment. The employee's first contact will be with the physician, who need not be on a full time basis. In smaller firms the examination can be made at the office of the appointed examiner. Following a complete physical examination, the employee talks with the physician, who should explain any defects and refer him to his family physician. He should be told that the findings of the examination are confidential. This is probably his first real physical examination. Perhaps he thought himself in perfect health and is grateful to know of his weaknesses, grateful to know that he is being accepted, and grateful for the opportunity to remedy his defects.

The purpose of a pre-employment examination is not to reject those with physical defects, but to place men in work for which they are physically fit. Rejects at Caterpillar are limited to those who have a contagious or communicable disease, or are in such physical condition that employment would be dangerous to themselves or others. Even those with a venereal disease are employed, providing the disease is not in a contagious stage and providing there is no evidence of neurological findings. Industry must accept its share of less than perfect applicants and strive to make them useful employees.

Pre-employment examinations alone fail to make a health program. Periodical examinations at stated intervals or examinations made at the request of the employee or supervisor are of great value. Many times we have saved an individual from weeks of suffering and perhaps death because the supervisor noted a change in the employee and sent him to the physician, only to find the insidious on-set of a disease which, if allowed to progress, would have resulted in many weeks of lost time.

Key men, whether they be two or five-hundred should be examined yearly. The safe guarding of their health is quite obvious, for the replacement of these men often require years of training and much expense.

There is consolation to an employee who knows that he is working in an industry where medical care is always available. He relies upon medical attention and learns the importance of prompt and expert care. He realizes from his experiences at the time of examination that the physician is interested in him as an individual and that his ailments are retained in strict confidence. He finds himself drawn more closely to the doctor, finds himself more confident in his ability and more eager to seek his advice and guidance. It is apparent, especially at the beginning of a health program, that men are suspicious, but they are soon convinced that the program is for their benefit and not some sinister scheme to weed out those who fail to pass as perfect physical specimens.

The services of a physician and nurse are not the sole requirements of an effective program, but only one phase. The other phases involve working conditions, heat and ventilation, production standards, sanitary toilet facilities, luncheon facilities and many factors too numerous to mention but which nevertheless directly influence the maintenance of health. The most perfect medical set-up possible is valueless without the sincere, active and intelligent participation of the entire management in the program.

HEALTH programs in the past have often been considered a non-productive portion of business. Nothing is farther from the truth. This is evidenced by facts which are undeniably true to everyone with experience in industrial medicine. The following is a statement from the president of Caterpillar Tractor Company: "Consider, for a moment, the financial side of an industrial health program. Any program that benefits health cannot help but benefit in many other ways as well. Let's consider just a few of these other benefits—the most obvious ones.

THERE IS LESS LOST TIME—Men learn the value of preventive medicine and when distressed are quicker to seek medical attention. Men with contagious diseases are kept home, and by prompt action epidemics are curbed. There are fewer illnesses because health is guarded. As a result, there is less lost time. Lost time, of course, costs both the employee and the employer money, and a diminishing of lost time represents a real saving.

THERE ARE REDUCED ACCIDENTS—Men who are not in good physical condition are the most likely accident candidates. They are less alert, their reactions are slower, they are less willing to take precautions, and their weakened condition in itself is hazardous. Accidents are expensive, and a reduction in them not only reduces human suffering but also reduces costs.

THERE IS GREATER EFFICIENCY AND COOPERATION—Certainly, improved health also means improved work. Health has a direct bearing upon attitude and performance, as we all know, and as health standards go up the degree of cooperation likewise goes up. The man who is in poor physical condition is neither likely to do good work nor get along well with the other men. He tends to be indifferent, temperamental, tactless and dissatisfied. His physical condition may not only affect his own work but the work of many others. So here again a health program pays substantial dividends.

There are other, less tangible, benefits which are also worthy of mention. As well as building healthy men, an industrial program builds healthy good will, and very definitely improves employer-employee relationships. I am sure that the more time a firm devotes to a medical program the less time it will have to devote to misunderstandings.

The benefits of a health program have been considered in their relation to employer and employee but certainly added to it there are the influences on the health standards of a community. We definitely feel that by influencing the health of our 13,000 employees, we have contributed much to the health standards of our community. If every smaller industry would join in the program,

humanity would benefit in an incalculable measure.

In summing this phase of an industrial program, the following by Frank R. Peregrine is appropriate: "The fact that industrial health work pays for itself in dollars and cents (of which there can be no doubt) is by no means the main reason why it should be fostered, amplified, and carried on by industry. Happy can be and should be the industrialist who, in the declining years of his life, when reflections come, and when the words of that old hymn recur more and more,—Just a few more years in this vale of tears, and the story will be ended,—can conclude his reflections upon the achievements of his life in the field of industry with the thought: And this I have done; this I have accomplished; this I have achieved without harming the health or shortening the life of any fellow man who helped me do it.

IF THE facts above set forth are acceptable and the need for an industrial health program is admitted, then consideration must be given to proper and adequate equipment. This requires considerable thought and planning. The site selected must be so situated as to prevent unnecessary lost time in going and coming from the point of employment, and often it is economical to have more than one first aid station, as the conservation of lost time can more than offset the cost of operation. The space selected should be large enough to accommodate the needs of the industry involved and should be equipped with sufficient natural light and ventilation, and so placed for the maximum degree of cleanliness. The medical equipment and arrangement should always be under the direction of the physician with the nurse or trained first aid personnel reporting directly to him. The organization should be in close relation to personnel, employment, safety and insurance, and, to be successful, there must be unity between all of these branches.

The equipment required depends upon the size of the industry and the number of individuals employed. Cheap and inadequate equipment becomes a severe burden in one or two years, while good surgical supplies will be standard equipment viewed with pride for a long time. The buying of equipment requires the good judgment of the physician with the advice of experts. To my knowledge there is only one surgical supply company today that has been sufficiently alert and interested to train men who may be called in consultation to discuss the needs and amount of equipment necessary.

These men, as the physicians are pioneering the industrial field, should be complimented and encouraged in their endeavors.

In conclusion, it is admitted that many phases of this great problem have not been adequately considered but at least the facts are indisputable. If we desire to be humanitarians by helping our fellow men and our community, as well as ourselves, then we must admit that a health program is a necessity for industries, large and small.

Current Comment

Abstract by

Emery R. Hayhurst, M.D., Ph.D.,
Columbus, Ohio

Silicosis in Danish Industry: Commentaries by Dr. M. Conrozier

EXPLANATORY NOTE: Silicosis in Danish Industry, by Dr. V. Stub-Christensen, is described as comprising a Thesis of 312 pages prepared in connection with the acquisition of a degree of Doctor of Medicine, and covering a three-year study which ended in 1932. It was later published as a textbook in the Danish language (Levin & Munksgaard, Copenhagen, 1937). This work is briefly reviewed under Book Notices in *JAMA* (112, 21:2200 May 27, 1939). No translation has yet appeared in English. However, a French translation of its summaries, rendered by Dr. R. de Bovis of the School of Medicine of Reims, appears in *La Medicine du Travail* (12: 12-37, January-March, 1940) followed by the *Commentaries* (pp. 38-44), of Dr. M. Conrozier, of Alès, Ex-Interne of the Hospital of Lyons, who, as a radiologist, has had much experience with the hard rock coal miners of the Province of Gard in Southern France.

The following is a rather extended abstract of Dr. Conrozier's *Commentaries*, which were very kindly translated from *La Medicine du Travail* by Mrs. Marjorie B. Berry, of Chillicothe, Ohio, with only slight alterations by the abstractor.

DR. STUB-CHRISTENSEN'S very important statistical research confirms our own everyday findings that silicosis is a purely occupational disease. His extensive task included the observation of afflicted persons in numerous shops and workyards and under the numerous difficulties which are naturally germane to such investigations.

The workers were examined carefully by means of clinical, radiological and bacteriological procedures, carried out systematically for each one while endeavor was made to eliminate from this study all doubtful or disputable cases. Only workers in active service were examined, from which certainly numerous former workers were not included whom the dusts had forced into invalidism. Not only industries already recognized as dangerous, such as sandblasting, but those reputedly harmless, such as brick-makers and cement-makers, were covered.

Stub-Christensen saw that wherever a workman breathes silica-ladened dust he is liable to contract a pneumoconiosis (even where this dust is of slight silica content). Even with cement containing 20% of total silica and only 2% of SiO_2 (the remainder in the form of silicate), after 20 years numerous workers show a fibrosis, and after 40 years, all have diseased lungs. This is something both new and serious. It not only extends the field of the pneumoconioses but changes the nature of the controversy regarding the relation of tuberculosis with dust disease. While certain of these workers develop a typical nodular silicosis and severe dyspnea, what connection has this fibrosis, which, in differing degrees, overtakes all the workers, with tuberculosis? It did not enter Dr. Stub-Christensen's mind that in order to be a cement-maker it was first necessary to be tuberculous.

His findings resemble considerably the conclusions of the Belgian government's investigations in industries other than mines.

Radiographic Arguments

THE major importance of the x-ray in the diagnosis of disease from silica dust is greatly stressed, since the dusts give a characteristic appearance. Nodular fibrosis is rarely seen in ordinary practice, except among workers exposed to silica dust. In our own rock-miners we have numerous instances showing the whole gamut of nodulations in the matter of density and size, and those with gross nodules are legion.

DR. STUB-CHRISTENSEN, like all observers, has verified in certain of his workers the undeniable presence of pulmonary tuberculosis but he has proved that most often the tuberculosis did not make up all of the lesions shown on the plates. Far from it, he has observed in the majority of the plates, the *proximity*, and not the *superposition* of the silicotic and tuberculous shadows. Most often, one apex (very rarely both) displays the classic picture of cavity formation. Everywhere else silicosis is established without intermixture and the base and the entire opposite lung show only the classic nodulations.

This is a valuable discovery. Silicosis and tuberculosis *exist side by side without one being superimposed upon the other.* This constitutes the appearance of silico-tuberculosis, which we have observed many times in our own rock-workers who are beginning to show emaciation.

How interpret these cases? Exactly as DR. CHRISTENSEN did: It was a matter of silicotics in whom the occupational disease made a berth for tuberculosis. But the tuberculosis established itself in one region of the lung (usually the apex), and the rest of both lungs remained as pure silicosis. "The fibrosis which is the basic tissue of silicosis, protected the rest of the lung and acted as a barrier." We believe this circumstance, in which the two diseases exist side by side, without commingling, forms the most decisive illustration of the autonomy of dust disease. Let us conclude that these radiographic considerations afford two kinds of proof:

1. It seems established that each type of dust brings on a silicosis having its own particular radiographic image. Thus, sandblasters show silicosis with very fine, semi-dense granules, a condition also seen in moulders but possessed of less density. STUB-CHRISTENSEN found among sandstone cutters ganglionic shadows set in a calcified circlet. Grindstone dressers, on their part, frequently show contraction shadows which are not observed among the victims of scouring dust. The latter show all the aspects of nodulations most often dense, while polishers show nodulations of unequal size and scattered distribution.

2. STUB-CHRISTENSEN was able to verify, as has MORGAGNI, that certain cavitary shadows of silicotics did not correspond with those of tuberculosis, but showed a central necrosis in a fibrous mass — interesting evidence which one may associate with pure nodular silicosis showing necrosed centers.

Association between Silicosis and Tuberculosis

DR. CHRISTENSEN leaves no argument open on the origin of silicosis: "It is dust and nothing but dust." Tuberculosis occurred among his patients in only two ways:

1. Either it had previously injured the parenchyma, lymphnodes, or pleura, especially along the fissures, and had completely enveloped the primary silicotic picture — a question in many cases of cicatricial shadows without clinical interpretation, — but in certain cases there was the impression (more clinical than radiographic) of its having aided in the more rapid evolution of the causes in a notoriously tuberculous community; or

2. The tuberculosis arose as a complication of a previously pure silicosis. This intervention was infrequent, and even rare in most of the industries studied by him, but it was notable that it hastened the evolution of many of his victims of scouring dusts.

DR. CONROZIER states that he has not a great deal to add to these findings of his Danish confrere. He says, we see a certain number of our miners spitting bacilli at a certain stage in their evolution, which seems to accelerate this evolution. Rarely do we see films showing cicatrices of old, healed tuberculosis. In fact, we have no clear notion that certain progressive cases of silicosis were previously dominantly tuberculous cases. "Our most numerous cases of rapid evolution belong to pure nodular or massive forms. Silicosis is often sufficient to bring the patient rapidly to his death without any other morbid factor."

Regarding the arguments which DR. CHRISTENSEN's work bring to the debate between tuberculosis and silicosis, "One of the first is that, in the very great majority, his patients remain pure silicotics during their whole evolution. It is true that tuberculosis is rather rare in Denmark. But in spite of the rareness of it, the author, who has for each of his cases inquired into the tuberculous morbidity of the individual and of his family connections, emphasizes the fact that, even in the case of workers with a tuberculous heredity, silicosis often evolves as a pure disease. Among the 11 polishers with silicosis in whose cases DR. CHRISTENSEN had discovered tuberculous antecedents, only one developed into silico-tuberculosis. This evidence is truly astonishing."

Tuberculosis was, on the other hand, quite frequent among silicotics in the scouring powder industry and DR. CHRISTENSEN thinks that this dust, by its own lesions, provokes a condition favorable to the onset of tuberculous complications. But he thinks likewise that the cases which develop most rapidly are encountered in those predisposed by their heredity. DR. CONROZIER refrains from taking issue with this, limiting himself to stressing that the majority of the patients were women and that the work was carried on under very bad hygienic conditions.

It must be recalled that the radiological images vary with the industry, and thus with each kind of dust. Such may depend upon the content of silica (or silicic acid) of each dust, and, in certain cases, upon the volume and form of the particles, which traumatize the alveoli more easily and thus prepare the ground for tuberculosis.

Whether there is a close connection between the intervention of tuberculosis and the physical constitution of the subject, DR. CHRISTENSEN has not been able to come to a definite conclusion, this remains a disturbing factor.

Finally, there remains the question of the pneumothoraces found among workers in dusts. DR. STUB-CHRISTENSEN's patients conformed to the rule that none of these curious cases evolved fatally, as in the tuberculous: none had the pleura infected, and none died with pus in the pleural cavity.

While apologizing at somewhat great length for splitting hairs about the work of the eminent Danish confrere, DR. CONROZIER thinks it still not useless to point out that this remarkable work does not bring forward the least argument for the thesis of the supporters of tuberculosis. "All the facts observed, all without exception, give evidence in favor of the solely occupational origin of the malady."

Conclusion

AS DOCUMENTS are gradually amassed, as researchers gradually apply themselves to extending their investigations to new industries, one reality is forced upon us: Silicosis is the most frequent and the most serious of occupational diseases. It is found in every place where the dust contains — even to a slight degree — silica. "The author found it among cement-makers and tile-makers. And the prodigious statistics which he cites of porcelain-makers, none of whom is indemnified if he reaches old age, bear witness to the very great importance of the question.

"Yet sandblasters, stone-cutters, polishers, porcelain-makers, etc., form, in the army of silicotics scattered throughout the world, only a numerically insignificant factor. The great part of this army is recruited underground: it is made up of the rock-miners of all countries.

"How many are there who suffer and die through dusts?

"Man's science has bent itself to good purpose in preserving humanity from great epidemic plagues. It ought now to devote this great work of preservation to its own victims. There is not, in social pathology, a more just or more urgent task than the struggle against dusts. It has sufficed for fortunate Denmark that DR. STUB-CHRISTENSEN has pointed out the danger of silica, for the country has decided that its victims shall have compensation. . . ."

Industrial Medicine
Reg. U. S. Pat. Off.

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Paradoxy

IN THESE days of war and preparations for war, one wonders! And wondering, one becomes another of the innumerables who are wondering now, and, in similar times, have wondered in the past. Most must wonder inarticulately; once in a while, however, an inspiration to expression of the wondering evolves a permanent paragraph. The following, quoted by Air Hygiene Foundation in a recent bulletin, is one such:

"Two contrary laws seem to be wrestling with each other nowadays; the one a law of blood and death ever imagining new means of destruction and forcing nations to be constantly ready for the battlefield — the other, a law of peace, work and health, ever evolving new means of delivering man from the scourges which beset him. The one seeks violent conquests; the other, the relief of humanity. The latter places one human life above any victory; while the former would sacrifice hundreds of thousands of lives to the ambition of one. The law of which we are the instruments seeks, even in the midst of carnage, to cure the sanguinary ills of the law of war; the treatment inspired by our antiseptic methods may preserve thousands of soldiers. Which of these two laws will ultimately prevail, God alone knows. But we may assert that science will have tried, by obeying the law of humanity, to extend the frontiers of life."

This was said by Louis Pasteur—who said much more that "Time shall never edit." In view of the place of the science of industrial medicine in the preparation for war, it has a peculiar significance.

Industrial medicine is, very aptly, the medicine of "work and health"; it is the law and the science of the maximum adaptation of each to the other and the happiest interdependence of both. At present the maximum of expression of both is the vital basis of readiness for the battlefield; the industrial physicians are as important in their part, as the captains and the kings in theirs. But — one wonders, and one hopes! The time will come, it must come, when the law of peace will prevail. And when it does, the law and the science of work and health will, indeed, "extend the frontiers of life" on *all* fronts — for there will be no sanguinary front of the ills of war. If it is necessary for a while to sustain the paradox of the utmost efficiencies in labor and well-being having as their highest purpose the speedy and uninterrupted preparation of the agencies of death, — so be it! That way lies the restoration of peace, through the victorious eventualities of being compelled to fight for it.

THE picture, however, has a brighter side, and, strangely, it is another paradox. The exigencies of war are developing to near-perfection something which will persist after the war; and, although in a sense it will be a result of the war, it will not be in any respect a salvage from it. The mutual adaptation and interdependence of work and health so essential in the arts of war can be transferred over intact to the arts of peace. Of what else can that be said? The armies will be mustered out, and the generals retired — but not the industrial physicians. They are engaged in an enterprise which is its own justification, whatever the direction of its aims from time to time.

And they will continue that enterprise beyond all changes in the frontier of their effort. *They* will be the same in the one as in the other — but in the latter it will be not only the law and the science of work and health, but, as the quoted paragraph indicates, the science and law of "peace, work and health."

The industrial physicians thus have an opportunity so tremendous that it is difficult to gauge its magnificence at close range. It needs the perspective of time to be appreciated. Not interested, however, in its historical future, they are rising to its immediacy in the manner of men accustomed to big things.

But it should at all times be remembered that they are *rising* because they have a place to rise from. The defense needs are not *creating* industrial medicine; they are merely demanding from it quickly and on a huge scale the expression of the experience and ability its pioneers developed when their frontier was everything from indifference to criticism. And thus they are rising, not merely to an emergency, but to a height from which all the frontiers of life will be forever visible. In time of war they are building for the time of peace, and the effectiveness of that peace in the future is inherent in the quality of what they are doing now.

INDUSTRIAL HEALTH AND DISEASE

Should Coronary Disease and Hypertension be a Cause for Rejection in Industry?

H. M. F. BEHNEMAN, M.D.,
San Francisco

THE answer to the highly controversial question "Should coronary disease and hypertension be a cause for rejection in industry?" depends to a great extent on who is asked—the injured worker, the employer, the trade union, the insurance carrier or the industrial physician. These are the people primarily involved in the solution of one of the most frequent yet most complicated problems confronting the student of industrial medicine. I use the word "student" because I know of no other pathologic problem in this specialty requiring more careful study in each individual case if the problem is in any way concerned with industrial liability.

High in morbidity and highest in mortality, heart disease is the most expensive illness. More than a fifth of cardiac disorders occurs in persons under the age of 40, and it costs this country $250,000,000 a year in lost wages, in addition to the cost of physical and financial dependence of persons with these disorders on the community and on industry. Furthermore, heart disease has a depressing and disintegrating effect within the family in which it occurs.

The compensation law of California specifically renders industry liable for "injuries arising out of or in the course of employment." High courts in other states have repeatedly decided that even a cardiac accident arising in the usual course of employment is compensable. Of the five interested parties mentioned previously the first four are inherently prejudiced from the start; therefore the physician qualified to review cases of cardiac disability in industry must be honest in order to be fair to all parties; he must be scientific to be thorough, in that he must have sound physiologic understanding of each case before he can come to a rational conclusion. The worker disabled by heart disease too often, but naturally, ascribes it to trauma or activity in his occupation. The employer is interested in efficiency, regular hours, speed and mass production. The trade union is concerned with the care and protection of its members. The insurance carrier wants to appease its assured yet show a profit for its stockholders without raising the employer's premium. Into this situation walks the physician employed by any of the previously mentioned parties to render an opinion or give expert testimony before a referee, judge or jury. The less said about the present status of so-called expert testimony, the better; what is needed is the creation of standards for qualification, to be defined and approved by physicians in each specialty in medicine.

The answer, then, to the question of industry's true course in the rejection of workers with heart disease and those in the hypertensive state can lie only in the merits of each such worker. Moreover, the answer rests on whether or not the applicant is applying for his first job with an employer or whether he has become disabled during the course of employment and reapplies for his job.

To any one with experience in the field of industrial medicine it is utterly absurd, and unfair to all concerned, to attempt any blanket rejection of men willing to work just

Read before the Section on Preventive and Industrial Medicine and Public Health at the Ninety-First Annual Session of the American Medical Association, New York, June 12, 1940. Published in *J.A.M.A.*, January 18, 1941.

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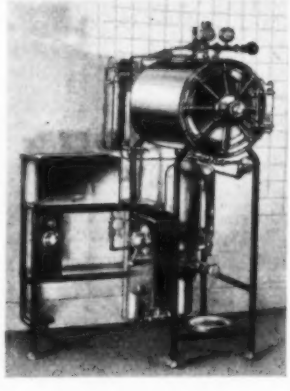

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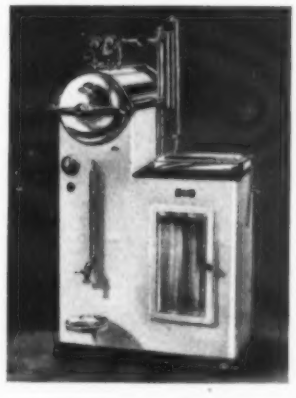

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because they have cardiac or circulatory disorders. There's a job for the man and a man for the job, without detriment to either.

Consider the applicant for work who desires his first job with an employer. His case is simply and easily handled. If on thorough examination the man is found to have heart disease or circulatory hypertension, his acceptance or rejection should depend solely on whether the work he is to do will increase that disorder or whether the disorder will reduce his efficiency in that job. If either of these factors exists, rejection is warranted and is logical and best for all concerned. The disposition of such rejections will be mentioned later.

On the other hand, rejection or re-employment of the worker disabled on his job is a problem of great magnitude, the solution of which rests on many interrelated factors. To me the answer is plain. Rejection or acceptance should depend on the data obtained from a scientific and comprehensive study of each case. With this, then, as my premise, let me briefly but factually state the underlying mechanisms concerned in the production or exacerbation of heart disease and hyptertension, on which, in turn, rejection hinges. But first a warning is in order. The physician in attendance must not assume the point of view of the private practioner. In the latter's study of a patient there always lurks an automatic consideration of heredity, environment, family history, habits, character and other factors which determine one's longevity. In industrial medicine these things are not forgotten, but the law reads that the worker is entitled to compensation for "injuries arising out of and during the course of employment." The referee, judge and jury are little concerned in the physician's knowledge of the injured worker's family history, his background, stamina or stability, which are potent factors in health and disease. They want to know "Was or was not this man's illness, disability or death a result of the work he was doing?" Most workers are not physically examined before employment; if they are, a signed waiver on physical defects does not always legally protect the employer. Remember that even the exacerbation of a preexisting illness gives the worker (as well as dependents of the deceased) the same benefits and rights as if the work created the disease state. This is manifestly unfair, but it is the existing law, at least in unionized California, land of the three bridges.

Consider, then, what progression of events in an occupation could create coronary disorders and hypertension or exacerbate them to the point where industry would be called on to prove or disprove its responsibility. To do that physicians must proceed to fundamental physiologic facts enabling them to render a rational, scientific opinion. My own views are based on experience in such cases and on the conclusions reached from animal experimentation. I firmly believe that industry has not been held liable for cardiac disorders and hypertension in a vast number of instances in which it has been partially or wholly at fault. The majority of medical opinion rendered in California is against industrial liability.

Next, I feel industry should not be held liable for coronary and vascular accidents occurring when a worker is performing the usual routine activities of his job, with no unusual effort or exertion or trauma. In that case, one rightfully feels the disorder was approaching and the inevitable symptoms and signs were soon to appear regardless of the worker's activities. However, when a worker's heart disease arises from his being subjected to trauma or activity bringing extraordinary or unusual effort in the course of his work it is difficult to see how his claim for compensation can be dismissed and his disease classed as of non-industrial origin without a comprehensive examination; and these are my reasons for such an opinion:

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the only supply of blood to the heart muscle. The heart's ability to increase its work depends on the oxygen supply. The minute anoxemia enters the picture the heart begins to fail. Heart failure is more pronounced in the hypertensive state, valvular disease and aortic abnormalities. The coronary blood flow depends on three factors: (1) aortic pressure, (2) mechanical resistance in the myocardium and (3) the caliber of the coronary vessels.

Arterial hypertension causes heart strain; it places a burden on the left ventricle, which develops the head of pressure for coronary flow. This ventricle can carry tremendous loads for years, but when it is partially anoxic its failure begins. When this occurs, however, in the right side of the heart there is far less ability of the right ventricle to carry on, with much faster failure when the pulmonary arterial pressure is increased. The speed and degree of failure, therefore, will depend on the unusual effort the heart is subjected to. Heart failure due to unusual effort is the more common type of acute or chronic myocardial failure. It is often produced in occupations which suddenly demand more exertion than the heart muscle can supply.

Now appears the question of industrial liability for death from coronary thrombi. Too often have I heard testimony to the effect that "Well, occlusion of this man's coronary vessels would have occurred soon, regardless of his work." It is true that the majority of published opinions have reported most coronary failures occur while the patient is at rest or asleep. But do not forget that hours to days usually elapse between the inception of a thrombus and its movement to occlusion. What right have physicians to say that inception took place at rest rather than at work, or that after inception the ensuing work did not speed up or increase the severity of the occlusion? After all, if a man labors five days at eight hours a day he is working 40 out of a 168 hours, which is less than a fourth of his week. Therefore his chances are three to one of having his attack when he's not working; but this fact provides no logical basis for excusing industry, for the hour of inception of a thrombus is all important.

Physical exertion and emotional states promote formation of thrombi, with resulting disaster. Again let me stress that if exertion has been the kind to which a worker has been usually exposed one should ascribe death to natural causes, but any degree beyond the usual, or exposure to toxic substances, temperature changes or oxygen deficiency warrants investigation of all circumstances when a cardiac accident occurs. Such an investigation must cover the period of time from the attack to a time at least a week previous to it.

Likewise, both physical exertion and highly emotional state produce hyptertension of varying duration. Thus high intracapillary pressure is produced. In the presence of marked sclerosis or other forms of circulatory degeneration the capillary vessels are prone to give way, with resulting hemorrhage which, if large enough, becomes a damaging infarct. Various observers have found severe physical exertion raises blood pressure from 100 to 180 mm. of mercury. If the rupturing capillary in itself does not produce interference in function, it initiates a train of events leading to future trouble.

Trauma of various kinds may also produce coronary closure, infarction and angina pectoris. I regret space does not permit the review of interesting and conclusive evidence. Penetrating and nonpenetrating blows to the chest and back have produced clearcut coronary disorders, the mechanism of production being due to vessel spasm. This may be transient, with little or no after-effects, or it may produce ischemia long enough to cause serious trouble. In workers buried in falling earth, increased intravascular pressure develops. Congenital defects may allow ordinary exertion but not unusual.

In other words, anything increasing the load of an inadequate heart muscle tends to produce heart failure, whether it be through coronary occlusion, spasm, rupture or aneurysm. Death may result from acute dilatation, as in electric shock, exposure to

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cold water and freezing temperatures. In the last war physicians saw the "nervous heart" with pain due to fatigue from persistent rapid beat. A worker with severe anemia cannot always respond to excess exertion without cardiac symptoms; in wounded patients the heart is affected by histamine shock; in those with burns, where blood is concentrated, there is diminished volume; in patients suffering hemorrhage there is an increased rate; in those with heartstroke death appears to occur from hemorrhage under the endocardium of the left ventricle. A heightened metabolism belabors the heart; diabetes accelerates sclerosis, and obesity accelerates coronary disease. A gain of 30 pounds (13.6 kg.) in weight adds nearly 20 miles of blood vessels to our vascular system.

If the cardiac output is increased a rising blood pressure increases coronary flow, thus subjecting the diseased vessel to rupture if the dilating response to epinephrine is insufficient. Also, increased cardiac output (per beat) tends to tear débris from disease valves as blood passes through, surging into the coronary openings of the aorta or into the general circulation. On the other hand, exertion may increase the circulatory rate without increasing the heart's output; then coronary pressure tends to fall and patients are in trouble, not from hemorrhage, but from anoxemia or clotting. Reversely, anoxemia in excess is a potent coronary vessel dilator. Why can't such dilatation in an atheromatous vessel be dangerous? Also an abrupt increase in the work of the heart or sudden lowering of pressure in the aorta starts a vicious circle which may result in coronary insufficiency. The resulting anoxemia causes a rapid circulatory rate resulting in an increase in the size of the right ventricle to a point where the all or none law does not apply—an increase leading to edema of the lungs; it is known that the right ventricle hasn't the reserve strength of the left one.

Moreover, body tissue and chemistry play a major role. On tissue metabolism depend the rate and volume output of the heart, which is merely an agent. The oxygen and carbon dioxide pressure, together with the hydrogen ion concentration in the capillaries, is the determining factor. During muscular work the circulation rate is considerably increased, both from the chemical side and from the fact that excessive breathing brings about a failure in circulation. Work increases oxygen consumption; when there isn't enough oxygen, through inability of the heart to produce it, the ensuing asphyxia impairs the action of the heart. Excess respiratory activity can cause shock, as forced breathing diminishes the circulation. This is particularly true in the brain, which has a rich blood supply for its gray matter. Its normal oxygen pressure is very high; during muscular activity oxygen diminishes and carbon dioxide increases because of the venous blood from the muscles. The role of excess lactic acid formation is questionable. It is known, however, that when tissues are poorly supplied with blood unoxidized lactic acid accumulates in them and passes thence into the blood stream, raising the hydrogen ion and carbon dioxide concentration of the blood and followed by stimulation of the respiratory center.

Thus it can be seen that any factor producing even mild failure of the heart embraces multiple and complex changes in the whole circulatory system.

SUMMARY AND CONCLUSIONS:

Augmented work increases oxygen consumption. When the entire heart is confronted with a general lack of oxygen the result is the picture of congestive failure; when there is a local lack of oxygen in the myocardium the result is angina pectoris, if mild, and infarction, if severe. The normal heart is usually able to meet the load placed on it by industry; there are few exceptions. Persons with abnormal hearts, circulatory hypertension and vascular diseases sometimes fulfil the demands made on them for a lifetime of activity but more often tend to fail eventually from even the routine activities and frequently fail when extraordinary activity is demanded. The presence of systemic disease is detrimental to normal circulatory function. Cardiac failure is in direct ratio to oxygen

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want. Exertion, disease and emotional states cause hypertension, which is usually a forerunner of coronary disease. The mechanism of production of coronary disease and hypertension has been reviewed in the light of their relation to industry.

Industry should reject workers with coronary disease and hypertension only when the work contemplated is clearly destined to exceed their ability to respond normally. Industry should accept responsibility for the pathologic conditions it has created or exacerbated. Industry and unions should cooperate by alloting work within the ability of the worker with heart disease, because 70% of those rejected are able to work at something. Differentiation between ordinary and extraordinary activity is difficult but possible.

In conclusion I offer the following thoughts toward solution of this controversial subject:

1. Institute pre-employment examinations with the right of the worker to sign a waiver which the law will recognize, thus acquainting the worker with his disabilities while protecting the employer.

2. In cases of questionable liability for cardiac disorders arising out of employment, demand careful medical analysis of each case by a competent examiner, who will painstakingly start his investigation not just from the time of onset of apparent disability but from a period many days previous thereto.

3. Establish wider use of electrocardiography, the value of which often exceeds that of the widely used roentgenography.

4. Amend the present industrial laws to allow finer gradations and degrees of incapacity.

5. Abolish the unfair laws extant in many states where a worker's death gives dependents full award when death has been due only to exacerbation of admitted, preexisting disease. Industry should rightfully reject a claim for full death benefits in the rupturing of an old syphilitic aneurysm at work which would have ruptured soon without any exertion; at present there is little or no allocation of degree of responsibility. In California now the award is $6,000 net.

6. Give the disabled worker prompt and proper medical care; rehabilitate him. Don't discard him, but develop him, and work with placement and rehabilitation bureaus which find work for disabled persons in skilled and unskilled labor.

7. Define more clearly in each state which occupational diseases are compensable and to what degree. As proposed by Robert T. Legge, create legislation enabling the person with heart disease to work under medical supervision yet releasing the employer and insurance carrier from financial liability in case of death.

8. At present, expert medical testimony is in a deplorable state. Create and define standards of qualification and urge their acceptance by commissions and courts.

9. Teach more industrial medicine in the nation's medical schools.

10. Create state medical boards of review which should decide which cases are worthy of consideration by commissions and courts. Such boards would eliminate many evils of the present system of decisions by lay referees, whose honest intentions are often buried in a mass of conflicting medical testimony.

Lastly, the principals must get together; the worker, the union, the employer, the insurer and the physician all have something to learn from one another.

That is my answer to the question before me. It cannot be a sweeping, conclusive one because it depends on the merits of the worker in each individual case.

This answer is a challenge to the industrial physician, especially when, with the vast defense program of the United States imminent, there will soon be a chance to place every available person. It is one I know will be met successfully to provide another brilliant chapter in the annals of American medicine.

Abstract of Discussion

DR. NELSON D. MORRIS, Toledo, Ohio: I agree with Dr. Behneman that present knowledge does not warrant widespread rejection of workers for hypertensive or coronary disease. Adoption of such practice to

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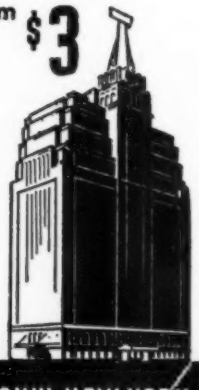
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protect the employer would implicitly force him to accept undue liability for those not rejected. Errors in courts or in legislative chambers in assigning undue risks to industry in re health of employees should not be allowed to stampede us into a false position. We have two important responsibilities: first, to safeguard and maintain health; second, to assist in determining causes in which injury attends occupation. Our first duty is performed by careful history taking, physical and laboratory examinations, reasonable effort to suit the task to the worker's capacity and continued supervision to find where we may be wrong, where physical status may change or where the conditions of labor may be improved. Industrial medicine so practiced aims to benefit every one through benefits conferred directly on the worker. It has no purpose to protect the employer from health risk except by keeping workers with known physical impairment from tasks of extra hazard and reducing those hazards wherever possible. The discharge of our second responsibility should not be confused by efforts to favor the interests of either worker or employer. Our obligation of impartiality is no less than that of the courts. If we give evidence or assume tasks unwarranted by our knowledge, we prejudice the decisions of judicial and legislative bodies. Unfair laws and unjust decisions reflect directly on members of our profession. Our duty is to know all that is knowable and to tell only what we know. We do not know the role of effort in creating or aggravating the condition in the vascular diseases under consideration. We can picture several possibilities for the victim of thrombosis beginning during sleep. He may die without waking, feel pain and call help or get up in the morning unaware of what has happened. His first symptom may then occur while dressing, on his way to work, while doing his usual task or soon or late during unusual exertion. Then who is to say what was the role of effort in disabling or killing this man? If a worker suffers death with infarction without thrombosis under conditions of labor that might be termed accidental, and if his collateral vessels are found normal, his death may be attributed to industrial accident. But preliminary examination would hardly have saved the employer from such risk. For the present, we can be more efficient in the prevention of accidents than in the selection of workers able to withstand them.

DR. ROBERT T. LEGGE, Berkeley, Calif.: The question as to who can work is today an important problem. The employer in many instances would prefer to employ rejected men who have heart disease but are prohibited by compensation laws, increased insurance rates and the fact that a waiver is illegal. The unions are opposed to the changing of laws for any special legislation, fearing that it may invalidate the workmen's compensation act. The attitude of industrial physicians is in accord with the health programs for workers for preemployment and periodic examinations. The purpose is, of course, to improve efficiency of the individuals and to detect early defects that may arise in the course of employment. The vocational guidance committees of the various heart associations are definitely in accord in their efforts to solve problems by some uniform methods of class legislation, by special waivers or by the subsequent injury act. Dr. Behneman states that in California compensation is given in heart cases. Germany has a permanent disability provision requiring employers to carry 2% of their injured. Such a measure should be adopted in our compensation laws. The National Institute for Cardiacs (Australia) not only secures employment for patients with heart disease but also guides and supervises them and establishes basic wages. America would be wise to adopt a like measure. Our knowledge today of coronary disease and hypertension warrants some modification of compensation laws in employing persons handicapped by heart disease, as the evidence here offered tends to substantiate. The Chicago Heart Association found that 1.34% in 13 industries were rejected on account of heart disease; 32.5% of these were found able to go back to active service, while 42.2% were restricted for modified duty. Master, Dack and Jaffe find only 2% of coronary disease the result of effort. Berghoff, of Chicago, states that the mortality of coronary block is less than 10%. Wychgel, in studies of arterial tension in steel workers 54 years of age,

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found that examinees with a systolic pressure of 200 and a diastolic pressure of 120 should be rejected.

DR. E. J. STIEGLITZ, Washington, D. C.: In connection with Dr. Behneman's paper I take some exception to the implied simplicity of the physiology of cardiac dynamics. I think that on further thought he would emphasize other factors, such as the availability of hemoglobin, the oxygen tension of the atmosphere, the availability of carbohydrate and insulin, which are all required by the myocardium. Furthermore, arterial constriction does not increase the pressure in the capillaries; it diminishes the blood supply distal to the narrowed arterioles. There is a definite slowing and sluggishness of the circulation distal to arteriolar constriction in hypertensive disease. A coronary accident, a coronary occlusion with or without infarction (and I have noticed the absence of any implication of a quantitative sense in evaluating coronary thrombosis and its consequences on myocardial function), is but a phase of the disease arteriosclerosis. The patient is not well prior to his coronary accident and doesn't necessarily become permanently disabled thereafter. The coronary occlusion is merely an acute exacerbation of a process which continues to exist and will continue to exist until death. It may or may not progress rapidly, depending on many factors. We must not separate too sharply a phase of the disorder from the disease itself. A similar thought applies in hypertensive arterial disease. We must recognize that it is a progressive matter. Neither hypertensive disease nor arteriosclerosis is curable. Therefore we should not talk about "cure" to the patient, the employer or the insurance company. These disorders frequently are, however, controllable. In our statements to these persons, be they patients or prospective employees, we should emphasize "control" of their disorder rather than "cure." It is not a question of being well or sick, as though there were a sharp line of distinction between the two. I doubt that any one of us in this room is 100 per cent well. The major problem in these cases is not the height of the arterial tension at the time the man is up for employment or reevaluation of his health but the rate at which his disorder is progressing. Determination of the rate involves somewhat more of a clinical examination than is carried out in many health surveys.

DR. ARTHUR M. MASTER, New York: I agree almost entirely with the suggestions made, but I should like to take issue with a statement that effort precipitates coronary occlusion. My associates and I have reported that from one half to one million attacks of coronary occlusion occur annually in this country. Hence it is important to determine whether effort can precipitate coronary occlusion or thrombosis. It will readily be admitted that effort can induce angina pectoris and failure of the left side of the heart with pulmonary edema and probably failure of the right side of the heart. However, there is no evidence that effort can precipitate a coronary occlusion. All persons exert themselves strenuously frequently during the day. Driving and parking a car, raising windows which do not give readily, are commonplace severe efforts performed repeatedly by most people. If effort were a factor in precipitating coronary occlusion, every one with coronary artery disease would have an occlusion. Furthermore, the latter occurs in all classes irrespective of occupation, again evidence that physical exertion is no factor in the formation of coronary occlusion. Recently it has been theorized that exertion increases the aortic pressure and thus ruptures a capillary in a coronary artery; this in turn is supposed to initiate a thrombus. There is no evidence for this. As Dr. Stieglitz says, a rise in arterial pressure is not transmitted to the peripheral capillaries. The area of the latter is so great that pressure falls precipitately. Again, Winternitz has shown that capillary rupture is part of the arteriosclerotic process, but nowhere does he intimate that effort or increase in blood pressure will rupture a capillary. In fact, Winternitz injected the coronary arteries of patients dying of coronary disease using the unheard of pressure of 500 to 1,000 mm. of mercury and yet did not produce capillary rupture!

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The Science, the Law and the Economics of Industrial Health

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The Toxicity of Antimony

—*Animal Studies*—

WILLIAM R. BRADLEY, M.S.,
and
WILLIAM G. FREDRICK, Sc.D.,
Bureau of Industrial Hygiene,
Department of Health,
Detroit, Michigan

LITTLE is known concerning the toxicity of metallic antimony and its compounds when used industrially.

Perhaps the first industrial employment of the metal dates back to the Chaldeans, who used it about 4000 B.C. in the manufacture of vessels and vases. Archeological discoveries show that other ancient civilizations made use of both the metal and the black sulfide. The history of antimony has been excellently reported in the comprehensive brochure by Habeck[1] (1936). Examination of the extensive literature on these materials and other antimony compounds clearly indicates their widespread industrial utilization at the present time.

In the United States, industrial workers are exposed to antimony in the mining of the ore, the smelting, refining and utilization of the metal, and the production and use of its compounds. The metal is employed especially as an alloy with other metals and in particular with lead, tin and copper. Nearly all industrial lead alloys contain antimony. Chief among these are bearing metals, type metals, storage battery grids and pewters. Antimony is a strategic metal for national defense, being incorporated in the manufacture of munitions, tools and machines. The development of domestic antimony sources, enlarging ore reserves and increasing domestic smelter capacities as a national defense measure has progressed rapidly in the last few years.

Industrial processes present the opportunity for worker absorption of antimony in the form of

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fumes and dust through the respiratory passages and in the digestive tract. The literature reports[2] that absorption may take place also through the skin. It is held by some authorities that the chief danger to workers with antimony alloys is from lead, arsenic or other metals which they contain. However, many cases of occupational illness attributable directly to the manufacture and use of antimony and its compounds as well as its alloys are reported in the literature together with specific clinical symptoms of poisoning in both the acute and chronic forms.

This paper presents, first, a brief historical survey of the pertinent literature, and second, laboratory studies on the toxicity of antimony and five of its compounds. The experimental work represents utilization of 357 small animals in the antimony study and 160 animals in a comparative lead study. Included in the comparative work is a study of the minimum fatal intraperitoneal dose for lead metal and five of its compounds. In the antimony phase of the work the minimum fatal intraperitoneal dose was determined for rats, together with the resultant pathology in the fatal episode and in sub-lethal dose survivors. Rats and rabbits were fed antimony metal and tartar emetic for periods up to 12 months. Minimum fatal dose determinations were made of the metal and antimony potassium tartrate by mouth in rats and intraperitoneally and by mouth in guinea pigs. Hematological, gross and microscopic pathological examinations, chemical and spectrographic analyses were made on representative animals. The subjection of small animals to the inhalation of antimony dusts and fumes has, up to now, not been undertaken.

Historical

CAROZZI[2] in 1930, White[3] in 1934 and McNally[4] in 1937 have presented literature summaries of many industrial antimony poisoning cases together with clinical findings dating from the first description of the effects of antimony on chemists by Ramazzini[5] about 1700. Included in these reports are the findings of Schrumpf and Zabel,[6] 1910. They noticed among younger workers in a type foundry very few cases of lead poisoning, but a number with: "remarkable facial expression, complaints of nervousness, irritability, sleeplessness, fatigue, dizziness, headache, both frontal and cerebral, muscular pain, neuralgia in the extremitiesties, nausea, loss of appetite, gastro-intestinal disturbances and constipation." Examination of the blood revealed a diminished leucocyte count and a notable eosinophilia. A lowering of the blood pressure was noted. Antimony was found in the fecal discharges. The symptoms disappeared after suspension of work for two to three weeks. Experimental feeding of rabbits with antimony oxide and sulfide produced similar changes in the blood.

Selisky[7] in 1928 reported 200 cases of industrial skin lesions due to the use of a solution of antimony salts as a mordant in cloth dyeing. He described a pustular necrotic dermatitis commencing as a folliculitis and ending with atrophic scarring and cites intermediate products of the antimony salts as the etiologic factor. He further noted the condition to be worse in the summer months. Oliver[8] (1933) found among antimony trioxide workers occasional skin irritation, a slight lowering of the blood pressure and a marked daily excretion of antimony in the feces, but concludes that the occupation has little danger.

However, Feil[9] in 1939 reported considerable illness among workers in an antimony foundry. He states, "among 15 workers exposed to vapors (the white oxide and metal vapors produced in the melting process) seven men had a rash when examined and 14 had had it at some time. It consisted of pustules very similar to smallpox. The neck, forearms and legs were usually affected. In some persons the rash covered almost the whole body, while in others there were few pustules. They arose after a few weeks' work and healed when the worker left the job. The only worker who had never had the rash suffered greatly, however, from the vapors (lack of appetite, headache, oppression). Half of the workers had conjunctivitis and half had anemia and other complaints."

Factory inspection reports from Hamburg, Germany, showed that the workers grinding metallic antimony frequently suffered from dyspepsia, headache, vomiting, conjunctivitis and a bloody discharge from the nose. Dutch inspectors in Holland found what they held to be acute antimony poisoning in a man who broke up large pieces of the metal. It nevertheless appears that the opinion of authorities[2] is divided on the possibility that antimony may cause industrial poisoning.

A few toxicological studies dealing with industrially used antimony compounds have been reported. In 1912, Boveri[10] fed rabbits metallic antimony suspended in oil in doses from five to 55 milligrams every other day for 30, 60, and 90 days without apparent bad effects. Larger doses produced diarrhea with a progressive cachexia and death. Abortions were frequent. Autopsies revealed hypertrophy of the heart and adrenals. The aorta and other arteries showed no alterations such as are found in lead poisoning.

Seitz[11,12] in 1923 and 1924 corroborated his blood findings among type founders with those he obtained experimentally through subcutaneous injections of antimony in cats. A diminution in red and white blood cells resulted. Antimony administered by stomach caused a diminution of white cells and an increase in red cells. The results of experiments on guinea pigs were not so pronounced.

Manson[13] in 1926 conducted electro-cardiographic experiments following intravenous injections of antimony at doses of one-tenth to one-half minimum fatal doses. A slowing of rate, lengthening of atrio-ventricular conduction and arrythmia resulted and he concluded that the fundamental function of the heart muscle was affected and the action was local in the myocardial tissue and not in the inhibitory centers. A marked lowering of blood pressure also occurred.

Maneghetti[14] in 1926 injected trivalent antimony

(colloidal antimony sulphide and tartar emetic) slowly into the veins of rabbits. This caused a "tone blood disease" characterized by the appearance in the circulation, within 24 hours of numerous normoblasts, polychromatic erythrocytes, Howell-Jolly bodies, red cells with basophilic stippling and an increase in reticulated erythrocytes. There was leucocytosis with the presence of myelocytes. The red blood cell count and hemoglobin remained normal. After 200 hours, there was a lowered red blood cell count, a drop in hemoglobin and intense anisocytosis with many large normocytes. Normoblasts were also present. The rapidity of injection had a marked effect on the intensity of the blood changes, the degree of toxicity and the localization and nature of pathological changes.

Flury[15] (1927) conducted feeding experiments on rats, cats and dogs using tartar emetic, antimony trioxide and antimony pentoxide. He found that doses of the trioxide and pentoxide as high as four milligrams daily could be fed to 150 gram rats without appreciable effect on growth. Daily doses of 200 milligrams of the trioxide resulted in slight irregularities in the growth of rats while death resulted with the same dose of tartar emetic. Two cats sickened and lost weight after the continued feeding of these oxides at 450 milligram doses daily. The pentoxide was found to be slightly less toxic than the trioxide. Dogs were more sensitive than small rodents and showed digestive upsets, vomiting and diarrhea after the administration of only a few grams of material. Tartar emetic was found to be by far the most toxic of the compounds studied. About 10 milligrams per kilogram caused vomiting in cats and four milligrams per kilogram produced similar effects in dogs. Continued feeding of small amounts of antimony compounds including tartar emetic hindered the growth of young rats; with larger doses definite retardation of growth occurred. No certain evidence of tolerance to antimony was obtained.

In 1926, Pribyl[16] found an increase in the nonprotein nitrogen of the blood in the experimental subacute tartar emetic poisoning of four rabbits. This he believed is due to a rise in urea nitrogen and is associated with an increase in this substance in the urine. The ammonia nitrogen quotient in the urine of poisoned rabbits seems to be inversely proportional to the urea nitrogen quotient. The acceleration of protein metabolism is attributed mainly to increased autolysis of tissues.

Lucia and Brown [17] in 1934 injected a freshly prepared 1% solution of tartar emetic intravenously in rabbits. A six-milligram per kilogram dose reduced the leucocyte count by 50% within five minutes. No subsequent leucocyte loss was observed in 28 hours. Normoblasts were moderately increased while the blood platelet count remained unchanged.

Habeck[1] in 1936 reported a marked lowering of the leucocyte count in chronic antimony poisoning.

Oelkers[18] who in 1937 discussed the pharmacology of antimony, states that it is a general cell poison whose action disturbs "vascular breathing," checks albumen putrefaction and fat and carbohydrate metabolism. He further states that the view held by many that antimony causes capillary paralysis is not true. He found[19] that when rabbits were poisoned with tartar emetic, a considerable disturbance in the catabolism of glucose and alcohol resulted.

In 1923 Brahmachari[20] observed that guinea pigs could not survive intramuscular injection of a glycerine solution of antimony trioxide in doses of 25 milligrams per kilogram but survived 20 milligram doses.

Carlson [21] found that 8% of the antimony in the crimson sulphide and about 3% of the antimony in the golden sulphide was soluble in gastric juice. He considered it possible that this solubility was sufficient to be a source of danger to men exposed to these dusts.

Bachem[22] has shown that chronic ingestion of antimony produces "serious degeneration of the internal organs" without establishing a tolerance.

Experimental

MINIMUM LETHAL INTRAPERITONEAL DOSE IN RATS: One hundred and forty nine albino rats were used to determine the minimum lethal intraperitoneal dose for antimony metal and five of its compounds frequently used in industrial practices. The relative toxicities of these materials were sought by implantation of known quantities in the peritoneal cavities of the animals. The materials were finely ground and passed through a 325-mesh sieve to effect a small particle size and thus aid their absorption within the body. The slightly soluble compounds were incorporated in fresh corn oil, the soluble compounds in distilled

TABLE I.

MINIMUM LETHAL DOSE FOR ANTIMONY COMPOUNDS ADMINISTERED INTRAPERITONEALLY TO RATS

Material		M.L.D./50 Milligrams of Antimony per 100 Grams Body Weight	Total Weight of Compound in Milligrams
Antimony potassium tartrate	$KSbOC_4H_4O_6 \cdot \frac{1}{2}H_2O$	1.1	3
Antimony metal	Sb	10.0	10
Antimony trisulfide	Sb_2S_3	100.0	139
Antimony pentasulfide	Sb_2S_5	150.0	247
Antimony trioxide	Sb_2O_3	325.0	389
Antimony pentoxide	Sb_2O_5	400.0	531
Controls, corn oil		Survived	3 ml.
Controls, distilled water		Survived	3 ml.
Controls, untreated		Survived	

water. Many metallic materials, when suspended in corn oil, catalyze its oxidation and produce organic acids which convert insoluble compounds to soluble ones. This effect increases the toxicity of the compound and leads to erroneous conclusions. Accordingly, only fresh suspensions of substances in fresh corn oil were used. All dosage was based upon the percentage of antimony in the compound in order to facilitate comparison with the toxicity of other metals and metal compounds. Three groups of control animals were established. One group received corn oil intraperitoneally, a second group distilled water, while a third group remained untreated.

In Table I the antimony compounds used are listed in the order of their toxicity. The minimum lethal dose presented in column two is the dose which killed 50% of the animals. Column three is a calculation from column two showing the corresponding entire weights of the compounds injected.

The animals represented by this phase of the study were adult, healthy albino rats which met standards for normalcy previously established for this strain. All laboratory animals received high caloric, high vitamin diets augmented by lettuce twice weekly. The materials used were of high purity, caution being observed that any arsenic content was negligible. Postmortem examinations were made of all animals to determine the cause of death in the acute episode and to show the response of body tissue to absorption of sub-lethal antimony doses. The latter group of animals were sacrificed at various periods from 15 to 70 days following injection. Tissues from representative animals were examined miscroscopically.

Animals dying within a few days after injection showed dyspnea, loss of weight, general weakness, loss of hair and evidence of myocardial insufficiency. Predominant physical signs in survivors were marked immediate loss of weight with a retarded, gradual gain after five to 10 days and a marked loss of hair with a dry and scaly appearance of the skin. Hematological studies made of these animals showed an eosinophilia.

Pathological Findings

AT autopsy, gross findings in the acute episode were myocardial congestion with engorgement of cardiac blood vessels, dilatation of the right heart and little change in the lungs. The abdominal viscera were generally softened and congested with occasional interstitial hemorrhage. Numerous areas of the peritoneal surfaces were overlain with the injected material. Death was attributed to myocardial failure. In surviving animals the initial acute congestion had partially subsided when seen at autopsy, although cut sections of the hearts, livers and kidneys showed congestion and occasional signs of early degenerative changes. The small intestines usually were hemorrhagic. A fibrous tissue response in the abdominal cavity anchored peritoneal surfaces and walled off areas of the injected material in dependent portions.

Microscopic examination showed the lungs of all animals to be essentially negative. In those rats succumbing to lethal dosage, death was attributed to myocardial edema with marked hyperemia and capillary engorgement of the sub-endocardial zone. The livers showed moderate periportal congestion with vacuolization, degeneration and polymorphonuclear infiltration. Antimony oxides and sulfides produced no change in the spleen, but with the metal there was moderate hyperplasia with an increase in eosinophils. With the more soluble tartrate there was, in addition, an acute picture of intense splenic congestion. An acute toxic glomerulo nephritis (Fig. 1) with glomerular congestion, apparently albuminous material in the glomerular spaces and a degeneration of tubular epithelum also resulted in those animals receiving the tartrate. A similar response, but of mild degree, was found in rats receiving the lesser soluble materials.

In the microscopic examination of animals surviving sub-minimum lethal dosage more marked signs of chronic change were noticeable in the heart. A marked variation appeared in the staining of myocardial fibers. Intensely red staining cells, in which the fibrillar structure was indistinct, appeared more numerous in the sub-endocardial and epicardial zones. A fine stippling of dark staining pigment was present within numerous muscle fibers, more noticeable in animals receiving the metal (Fig. 2). Other myocardial cells were edematous. In the antimony potassium tartrate animals there was an increase in connective and fibrous tissues of the myocardium. In the livers (Fig. 3) of nearly all animals there was sub-capsular congestion and in some instances blood pigmentation. A mild hepato-toxemia was characterized by a functional hypertrophy of many cells displaying a cloudy appearance and finely

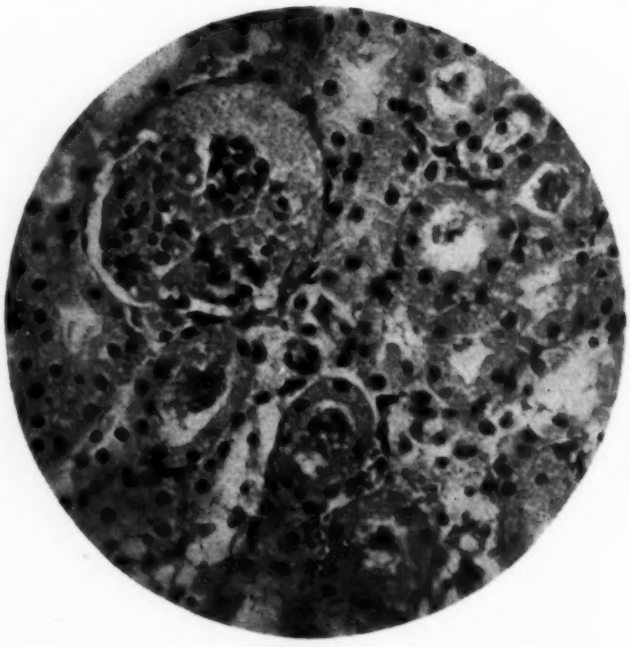

FIG. 1.
High power photomicrograph showing kidney damage characteristic of animals receiving lethal doses of antimony metal or its compounds

TABLE II.

Minimum Lethal Dose for Antimony Compounds Administered Intraperitoneally to Guinea Pigs

Material		M.L.D./50 Milligrams of Antimony per 100 grams Body Weight	Total Weight of Compounds in Milligrams
Antimony potassium tartrate	$KSbOC_4H_4O_6 \cdot \frac{1}{2}H_2O$	1.5	2.5
Antimony metal	Sb	15.0	15.0
Control—calcium carbonate	$CaCO_3$	Survived	100
Control—iron oxide	Fe_2O_3	Survived	100
Control—corn oil		Survived	2 ml.
Control—distilled water		Survived	2 ml.
Control—untreated		Survived	

reticulated cytoplasm. Numerous plasma cells and deep staining cells with picnotic nuclei were in evidence. In addition there was a slight periportal connective tissue increase with capillary congestion and infiltration of a few lymphocytes in livers of animals injected with antimony potassium tartrate. The spleens were essentially negative with a slight congestion and a moderate diffuse hyperplasia in animals receiving the metal and tartar emetic. Glomerular congestion with coagulated, albumen-like material in the tubules was also seen in the kidneys of these latter animals.

Comment

THE above findings establish a relatively low order of toxicity for the oxides and sulfides of antimony. The minimum fatal dose for the antimony sulfides, injected intraperitoneally, appears to be one-third that for the respective oxides. Antimony metal, however, presents a rather high order of toxicity. The high toxicity of tartar emetic, 10 times that of the metal, long has been recognized. The toxemia evident in the livers and kidneys is not surprising since such a response is frequently associated with the elimination of toxic materials. The suggestion here is that an albuminurea may be present during antimony absorption. The rather specific attack upon the myocardium is conspicuous by its nature and consistency. In the medical examination of workers exposed to antimony and to metals incorporating antimony, attention to physical signs and symptoms related to the heart may be justified.

Minimum Lethal Intraperitoneal Dose

THE minimum lethal intraperitoneal dose for antimony metal and antimony potassium tartrate was determined on normal guinea pigs. Approximately the same ratio of toxicity, 1 to 10, exists between these materials as when administered to rats (Table II). Control pigs received calcium carbonate and iron oxide in doses of 100 milligrams per 100 grams of body weight with the expectable absorptive and inert responses respectively. Additional control animals as listed in Table II were without poisoning. Eighty-eight guinea pigs were used in this phase of the study. Gross pathological findings in these antimony-treated animals was in close accord with those found in similarly treated rats.

FIG. 2.
Oil immersion photomicrograph showing fine pigmentation in myocardial tissue from a rat receiving a sub-lethal intraperitoneal dose of antimony metal

FIG. 3.
Low power photomicrograph showing liver damage characteristic of rats receiving sub-lethal intraperitoneal doses of antimony potassium tartrate

Comparative Study of Similar Lead Compounds

IN view of the fact that lead and antimony are closely associated in industrially used alloys, it appeared wise to compare certain similar lead compounds with antimony compounds. In this comparative study the minimum lethal dose of these lead compounds was determined by intraperitoneal injection into albino rats. Similar procedures were employed as when determining the minimum lethal dose for antimony compounds. One hundred and fifty animals received doses calculated as milligrams of lead present in the compound per 100 grams of animal body weight (Table III).

TABLE III.

MINIMUM LETHAL DOSE OF LEAD COMPOUNDS ADMINISTERED INTRAPERITONEALLY TO RATS

Material		M.L.D./50 Milligrams of Lead per 100 Grams Body Weight	Total Weight of Compound in Milligrams
Lead acetate	$Pb(C_2H_3O)2.3H_2O$	15	20
Lead oxide	PbO	40	43
Lead oxide	Pb_3O_4	45	49
Lead tartrate	$PbC_4H_4O_6$	70	99
Lead sulfide	PbS	160	181
Lead metal—325 mesh	Pb	above 100	above 100
Controls titanium dioxide	TiO_2	Survived	1500
Controls iron oxide	Fe_2O_3	Survived	1500
Controls calcium carbonate	$CaCO_3$	Survived	1500
Controls corn oil		Survived	2 ml.
Controls distilled water		Survived	2 ml.
Controls untreated		Survived	

Autopsy findings were those of acute lead poisoning. In these latter animals basophilic aggregation cell counts rose rapidly and gradually fell off or were lowering at the time of sacrifice. Control animals received 1500 milligram doses of titanium dioxide, iron oxide or calcium carbonate. Additional control animals are shown in the table. Basophilic aggregation cell counts in control animals showed averages in agreement with the findings of McCord and Bradley.[23]

In Table III the lead compounds used are listed in the order of their toxicity as indicated by minimum lethal doses. When this dose is used as the toxicological index, delayed action, cumulative effects or other responses which may occur will contribute to the order of toxicity. Fairhall, Sayers and Miller[24] found that lead metal and the lesser soluble compounds assume the same toxicological importance when the ability of the animal body to store lead was used as the criteria.

By comparing Table I and Table II it is evident that both antimony metal and antimony potassium tartrate are more toxic than is lead in the form of the soluble acetate when injected intraperitoneally into albino rats at minimum lethal doses. However, under similar conditions, lead as the oxide appears to be seven times as toxic as antimony trioxide. The minimum lethal dose for lead metal was not found. Doses as large as 100 milligrams per 100 grams of body weight did not kill the animals. The body weight remained stationary with this dose over a 40-day observation period. At autopsy, unabsorbed metal was found walled off in dependent abdominal areas or caught in the omentum. Antimony metal at one-tenth this dose was fatal to rats.

Oral Administration of Antimony

THE inauguration of a long continued feeding program, using the metal and antimony potassium tartrate, gave opportunity to observe not only the physiological response in small animals but also the distribution and retention of antimony throughout the body. The five phases of this program involved four series of albino rats and one series of rabbits, a total of 130 animals. Adequate controls were maintained for all animal groups.

The minimum lethal dose of antimony potassium tartrate by oral administration to rats was found to be 30 milligrams per 100 grams of body weight. Here again the dose given refers to the amount of antimony present in the compound. Rats survived 700 milligram doses of the metal but their weight remained stationary for many days thereafter. These materials were delivered directly to the stomach of the animals by rubber catheter and were incorporated in water and corn oil respectively.

In the second series rats were submitted to a feeding daily, except Sunday, of tablet triturates compounded in a lactose base from finely screened and thoroughly ball-milled ingredients. These animals were separated into two groups, one received treatment for six months, the other for 12 months. Tartar emetic doses were at one milligram and antimony metal doses at one or five milligrams. Feeding programs were begun on rats averaging 125 grams in weight.

In series three, rats were fed antimony ad libitum for seven and one-half months. Daily doses were one milligram for the tartrate and five milligrams for the metal.

Treatment of rats in series four was continued for 12 months. Doses of antimony potassium tartrate were gradually increased during the first six months to 10 milligrams daily per 100 grams body weight, at which level they were maintained. Similarly antimony metal was administered in gradually increasing doses up to 100 milligrams per 100 grams body weight daily. No tolerance was developed to these materials at the doses

used. This was demonstrated in a side experiment with rats where the highest doses were given without preliminary graduated step increases.

Rabbits were also given antimony ad libitum in daily doses of 40 milligrams of metal and eight milligrams of antimony potassium tartrate per kilogram of body weight. This dosage was continued four months.

Comment

ALL animals maintained their normal growth rates. Periodic basophilic aggregation cell counts showed normal rat averages of 2.0% and rabbit averages of 1. 8% throughout. Rats showed a slight leucocytosis. The gross and microscopic pathology of these animals simulates that previously cited for animals receving sub-lethal dosage intraperitoneally.

Oral Administration of Lead

THE response of rabbits given antimony differs from that elicited by lead compounds administered orally to similar animals. In a comparison study 10 rabbits each received five doses of lead compounds by stomach tube at intervals of four days. Doses were delivered in solution or suspension and the animals were submitted to autopsy from 40 to 90 days thereafter. The doses were computed in milligrams of lead present in the compound. On this basis, lead acetate, lead carbonate and lead oxide were given respectively in doses of 30, 200 and 200 milligrams per kilogram of body weight. The basophilic aggregation cell count in these leaded animals rose sharpely. Massive doses elevated this count to values as high as 15 times normal. At autopsy the gross picture was that of a typical lead poisoning.

Chemical Determination of Antimony

REPRESENTATIVE tissues from those animals receiving long continued oral administration of antimony were selected for chemical analyses. Various body tissues and organs including the brain, heart, lungs, liver, spleen, kidney, adrenals, bone, muscle and hair were used. The analytical method employed, using rhodamine B for the detection of small amounts of antimony, has been reported by one of us.[25] The tissue was wet ashed by treatment with hot 20% fuming sulfuric acid followed by 30% hydrogen peroxide. In 10 rabbits and 20 rats the predominant retention of antimony was in the heart, liver, kidney, lungs and bone. The hair also was found to contain slight amounts of antimony. Tissues of control animals showed the presence of antimony in amounts considerably less than in those from animals receiving antimony.

Analysis of 15 rat carcasses showed small amounts of antimony to be present. An average of one milligram was found in each carcass regardless of the daily dosage or the type of material administered. The carcasses of control animals each showed an average of one-tenth milligram of antimony as the normal constituent. Antimony apparently is not stored within the body as is lead. According to reports[2] it has been recovered in both the urine and feces following absorption.

Spectrographic Determination of Antimony

BODY tissues and organs from 34 representative animals receiving antimony by mouth were submitted to spectrographic determinations for antimony. The tissue was ashed and the antimony excited by a high voltage, high frequency, electrical discharge, essentially of the Tesla coil type. Pure copper electrodes were employed. The upper electrode consisted of a specially designed, inverted cone. The lower electrode was a plate. The tissue was placed on the lower plate and sparked until the sample was completely consumed. Standard samples were arranged by impregnating antimony-free tissue with known amounts of antimony compounds. Visual examination of spectrographic plates allowed a semiquantitative analysis to be made. The antimony line at 2311.5 ängstrom units was found to be satisfactory for quantitative work. A medium quartz spectrograph with a dispersion of 2 ängstrom units per millimeter in this region was employed. Eastman spectrograph analysis plates were used in this work.

Spectrographic determinations are in correlation with chemical determinations for antimony in various animal tissues and organs. High antimony retention corresponds to predominant pathological findings.

Summary

THE administration of common industrially used or produced forms of antimony to laboratory animals orally and intraperitoneally caused a toxic response which varied with chemical form as indicated by minimum lethal dose values. The most striking result was the high order of toxicity of the metal as compared with sulfides and of sulfides as compared with oxides.

The most important pathological effect was the consistent and invariable injury to the heart muscle, regardless of compound or dose in the concentration ranges studied. This injury was noted even at concentrations too low to produce diminution in the rate of animal growth.

Although chemical and spectrographic analysis of various body tissues of exposed animals always showed antimony in concentrations greater than those in control animals, the amount found appeared to be independent of the dose or the duration of exposure, and was never at a very high value. Apparently antimony is not stored to any extent in the organism.

Hematological findings were somewhat obscure and inconclusive. A slight eosinophilia was noted and no alteration of the basophilic aggregation cell count was produced.

Experimental comparison of the toxicology of antimony compounds with similar lead compounds showed no apparent similarity in the action of the two elements. In general antimony compounds are more toxic than similar lead compounds. Antimony, contrary to lead, appears to

be rapidly eliminated from the body without producing serious blood changes or suffering much storage.

Since all the antimony materials used in this study were of a high degree of purity, the primary effects noted could be due only to antimony. The possibility that the action might be due to lead, arsenic or other impurities present accordingly has been eliminated. In view of the definite toxic properties of antimony as shown by this and other work, its potential ability to produce occupational poisoning among exposed workers must not be disregarded. Signs of antimony intoxication in workers should be observable even when associated with lead absorption. Electrocardiographic heart studies upon antimony workers are definitely indicated.

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Industrial Mercury Vapor Detector

T. T. WOODSON,
Research Laboratory, General Electric Company, Schenectady, New York

MANY industries today utilize mercury or mercury compounds under conditions which may constitute a health hazard for the employees. The diversity of processes is shown by the fact that the manufacture of explosives, vacuum tubes, or even felt hats requires mercury, as well as do mercury boiler plants or petroleum laboratories. In all these places the mercury becomes exposed and possibly finely divided. Thus the vapor may easily be over the 0.25 mg/cu. meter toxic concentration estimated by Goodman in his excellent review of mercury poisoning.[1]

Methods made available for measuring atmospheric concentrations are two kinds — chemical or optical — and with few exceptions they are strictly for laboratory use.

Pioneer work in the development of a practical mercury detector was done in the General Electric Research Laboratory in 1926 by B. W. Nordlander.[2,3] He deposited activated selenium sulfide (SeS_2, a yellow powder) in a thin layer on paper. This layer darkened with the presence of mercury in the atmosphere above its surface, the reaction being a displacement of the selenium by the mercury, producing the black mercuric sulfide.

Since then, other chemical methods have been reported, but none have taken the place of SeS_2 in practice.

A partial list of present available chemical methods for detection of mercury in very small amounts is given.[4,11] In most of these cases the mercury is in the form of the liquid metal or one of its salts. Hence the methods are not directly applicable to the given problem.

More than 10 years ago Dr. C. W. Hewlett

developed a practical application of the optical method. This method is based upon the scattering of the "resonance" spectral line (2537A) by mercury vapor. Experiments showing this were reported by Wood about 1910.[12] This property of scattering can be demonstrated in a moderately dark room by placing a low pressure mercury vapor lamp 10 inches to 20 inches from a 6-inch x 6-inch ultraviolet fluorescent screen. The vapor from room temperature mercury, placed in a shallow dish between the two, appears as clouds of smoke on the screen.

Leighton and Leighton[13] have given an account of the optical methods, and they have shown unusual photographs of columns of vapor rising from a beaker of hot mercury, using visible and ultraviolet light.

This optical principle is applied to the detection of mercury in a transparent medium by directing the characteristic ultraviolet radiation through the medium to a phototube sensitive to 2537A radiation. The phototube yields a certain current in response to the ultraviolet radiation when the medium is free of mercury. The decrease of this response, all else being constant, means that mercury vapor is present, scattering the radiation to the walls.

A few writers have presented other or similar physical methods for the detection of mercury in the vapor state. Manley[14] gave a method for obtaining a spectrographic analysis. Hughes and Thomas[15] described a research on the absorption coefficients of mercury (from —15°C to +25°C) utilizing phototubes. This work was definitely of a laboratory type because the measuring instrument was a delicate electrometer.

Müller and Pringsheim[16] gave an application of this selective absorption. Theirs also was distinctly for laboratory use as they, too, employed an electrometer. This method was, however, for determining the mercury vapor content of air.

A recent Bulletin[17] of the N. Y. State Department of Labor has included a good summary of chemical and physical methods for estimating mercury in air.

Dr. Hewlett's detector was installed at the mercury boiler of the Hartford Electric Light Company, Hartford, Conn., where it still is in service. This installation was solely for testing flue gas and preventing the economic loss from a continued leak in the boiler. The health hazard in these mercury power plants is ordinarily low, but SeS_2 detectors are permanently placed at strategic locations for safety.

Improved Design

IN developing a simpler and cheaper detector for a power plant flue gas testing, it became apparent that a portable industrial model utilizing the same basic elements could easily be made. This newly developed model was designed primarily for electrical and mechanical simplicity. However, the characteristics of maximum stability against line voltage variations, ease of dis-assembly, high speed of indication and clearing, minimum maintenance, and great flexibility of application all were considered.

The result is a detector of the optical type using a small ultraviolet lamp, and one phototube, mounted in a welded housing of 2½ inch thin-wall steel tubing. The tubing forms the "absorption chamber" which is connected only by cable to the "control panel" which in turn supports the amplifier meter and power supply. A front view appears in Fig. 1. The circuit employed is a duplex triode bridge shown schematically in Fig. 2. The meter is adjusted to zero with clear air in the absorption chamber, the balance being effected by varying resistance.

Operation

THE introduction of a trace of mercury vapor into the chamber causes a reduction, in order, of transmitted light, photo-current, positive No. 1 grid voltage, and No. 1 plate current; and it causes an increase in No. 2 plate current and in the meter current. Thus the meter current measures the amount of light absorbed, and must reach a maximum when all the light is absorbed. This last condition can be created artificially by turning off the ultraviolet lamp. Since this maximum or "light out" meter reading is independent of lamp and phototube conditions (spacing, voltage, clean-

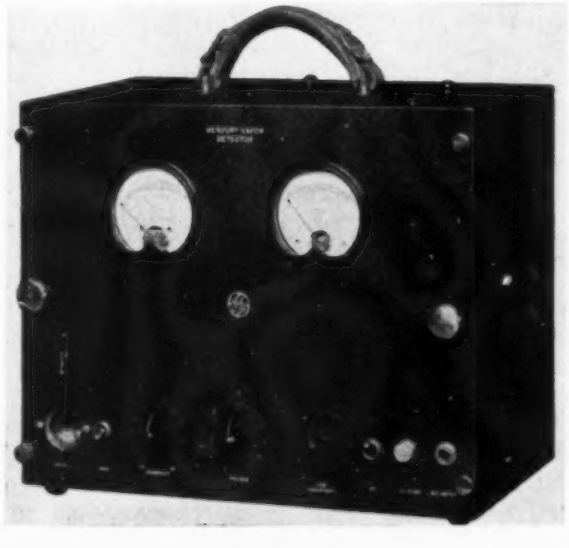

FIG. 1.
General Electric Portable Mercury-Vapor Detector

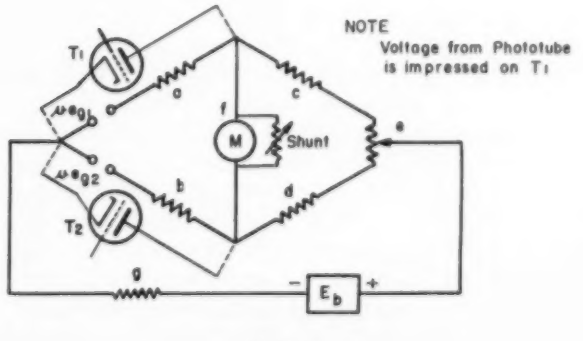

FIG. 2.
Systematic Circuit

liness, response, orientation), it is used as a basic setting. The device is calibrated by adjusting the meter to full scale on "light out" after balancing it to zero on fresh air.

Characteristics

FIG. 3 shows the approximate characteristics, all the family of curves being asymptotic to about full scale.

The detector under discussion, using a 10-inch chamber, has a basic calibrated range of about 0.5 to .005 part per million. Concentrations down to the one part per billion (.001 PPM) range can be measured if the sensitivity of the meter is increased by removing the meter shunt. This multiplies all readings by a factor of five. For higher concentrations the present detector can be used by diluting the specimen with fresh air in known proportions. Over 1.6 PPM cannot be measured at room temperature because the mercury will condense on the chamber walls and contaminate them.

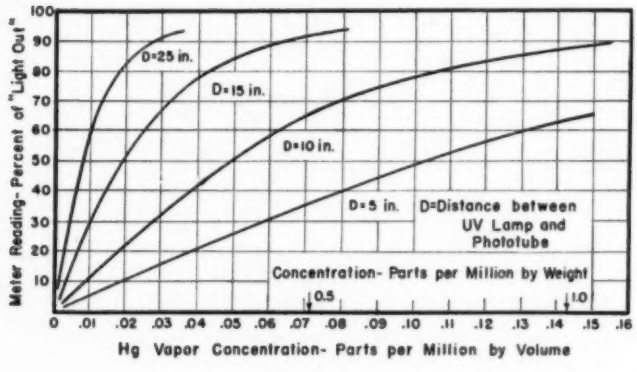

FIG. 3.
Approximate Concentration Characteristics

The mercury vapor can be carried by any gaseous medium which does not have a special absorption band overlying the 2537A region. Ozone does have such an absorption and hence affects the detector just like mercury, although to less degree. Likewise illuminating gas affects the detector, but only incomplete qualitative tests have been made with these two gases. From a group of 30 of the usual solvents, the only vapors to absorb the 2537A line significantly are benzene, pyridine, diethylacetal and toluene. Hydrogen, oxygen, ammonia, and nitrogen have been tried with negative results. Of course, smoke, fog, and dust act as physical light barriers and must be kept from the detection chamber.

The UV lamp like all low pressure arc lamps is sensitive to temperature so provision must be made to keep it within a range of ±25°F of an 80°F optimum. Also, the phototube must be kept below 150°F because the sodium coating will evaporate and cloud the window. A quartz windowed cell must be used for testing very hot or very cold gases.

The power requirement of the detector without the blower is about 60 watts. The net weight of the detector is about 25 lbs.; the panel is 11 inches x 14 inches, and the case is 7 inches deep.

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Ventilation for Electroplating

—New Data for Practical Design—

WILLIAM P. BATTISTA, THEODORE HATCH
and
LEONARD GREENBURG*

CHROMIUM plating creates a serious health hazard through the exposure of operators to the chromic acid mist dispersed from the electrolytic solution. Control of this hazard is commonly accomplished by means of lateral exhaust ventilation through slots extending along the two long sides of the plating tank. Suitable manifolds and ducts connect the two slots to the exhaust fan and discharge pipe. The rate of ventilation recommended by several investigators[1,2] to reduce the concentration of chromic acid to the safe limit is determined by the relation $Q = 100\ LW$, where Q = total rate of ventilation in cfm, L = length of tank in feet, and W = width of tank in feet.

It is clear that the rate of ventilation must vary with the tank width in such a way that a required minimum air velocity will be created at the center of the tank in all cases. According to the above equation, the relationship is taken to be a direct one; that is to say, the air velocity outward along the center line of the slot is assumed to vary inversely with the distance from the slot.

DallaValle's studies[3] of the velocity distribution around exhaust openings, however, have shown that the center line velocity in front of a suction opening is inversely proportional to an exponential value of the distance from the slot.

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*Division of Industrial Hygiene, New York State Department of Labor.

This exponent was found to have a value greater than unity. In view of this, some question may be raised with respect to the correctness of the above equation for tanks of different widths. The relationship has been investigated by the authors and the velocity contours and center line velocity equation obtained for a typical exhaust system on plating tanks. The results of this study are reported here.

In addition to the necessity for a proper relationship between rate of ventilation and tank width, it is clear that the air flow per unit of tank length should be reasonably constant throughout the entire length. This follows from the most elementary consideration of the ventilation requirements. It is not an easy matter, however, to obtain a uniform distribution of flow because space limitations often do not permit the use of the elaborate exhaust piping which is required. In practice, there is need for a simple and compact ventilating system which, with reasonable power cost, will provide a constant rate of ventilation for each foot of tank length. A second objective of the present study therefore was to develop the design requirements for such a system.

Two plating tanks of different widths may be said to have equally effective ventilation when the velocity contours over the centers of the two tanks have the same values. Hence, the rate of ventilation must increase with tank width in accordance with the relationship which governs the change in velocity with distance outward from the slot.

Ventilation Rate and Tank Width

THEORETICALLY, the velocity distribution may be considered from the standpoint of two limiting conditions: (1) a slot of infinite length or, in effect, a line source of suction of constant value throughout its length; and (2) a suction opening of such limited dimensions that the conditions approach those of a point source of suction. In the first case wherein an unobstructed line of equal suction throughout its length is assumed, the velocity contour surfaces will take the form of a series of concentric cylinders with the line source as an axis. Under this condition, the air velocity in the space around the line source will vary directly with the distance away from the line since the surface area of cylinders of equal length varies directly with their radii. Applying this to the ventilation of a plating tank, the following relation could be written:

$$Q = KLW \text{ or } Q/L = KW$$

where Q = air volume per unit time;
K = a constant;
L = tank length;
W = tank width.

This equation has the same form as the one now in use.

In the second limiting case wherein approximately a point source of suction in an unobstructed space is assumed, the velocity contour surfaces will take the form of a series of concentric spheres. The surface areas of these contours will vary with the square of their radii, thus:

$$A = KR^2$$

where A = surface area of spherical contour
R = radius of contour spheres

Considering R in terms of tank width, the relation between rate of ventilation and tank width becomes

$$Q/L = KW^2$$

The velocity distribution in the actual case of an exhaust slot on a plating tank does not conform to either of these limiting cases but lies somewhere between the two. It follows, therefore, that the exponent of W in the relation $Q/L = KW^a$ must have a value between 1 and 2. For tanks of a relatively great length, a will approach unity whereas for short tanks the exponent will have a value closer to 2.

DallaValle[3] has developed the following general equation for the relation between the center line velocity V at various distances X from the exhaust opening and the velocity, V_0 through the opening:

$$\frac{V}{V_0 - V} = KX^{-a}$$

A determination of the exact value of the exponent a for a typical exhaust slot was made by the authors on apparatus described later in this report (Arrangement *E* in Fig. 3).

Velocity measurements were taken by means of the thermal anemometer in a vertical plane normal to the midpoint of a horizontal slot 6 ft. long by 1 in. wide having an approximately uniform rate of air flow throughout its length. A smooth flat surface placed horizontally 7 in. below the slot simulated the electrolytic surface in a plating tank.

Velocity contour curves according to accepted procedure[3] corresponding to the common slot velocity of 2000 fpm for chromium plating are shown in Fig. 1. (These contours are for one slot only. For two slots on opposite sides of the tank, the combined contours would be equivalent to the vectorial sum of the contours shown in Fig. 1 and a reverse set of the same relative values established by the second slot.) Center line velocity values, outward from the slot, were obtained from these curves and plotted according to DallaValle's general equation in Fig. 2. The slope of the straight line resulting from this plot indicates a value of the exponent a of 1.625 for the 6 ft. slot. For other slot lengths the exponent will have different values between the limits of 1 and 2. In practice, however, plating tanks do not vary greatly in length and the exponential value of 1.6 may therefore be employed in the equation $Q/L = KW^{1.6}$ to determine the rate of ventilation in relation to tank width for most plating tank problems (NOTE—The velocity distribution characteristics of exhaust slots have been subjected to more elaborate dimensional analysis by Silverman,[4] the results of which will provide valuable information relative to the ventilation requirements

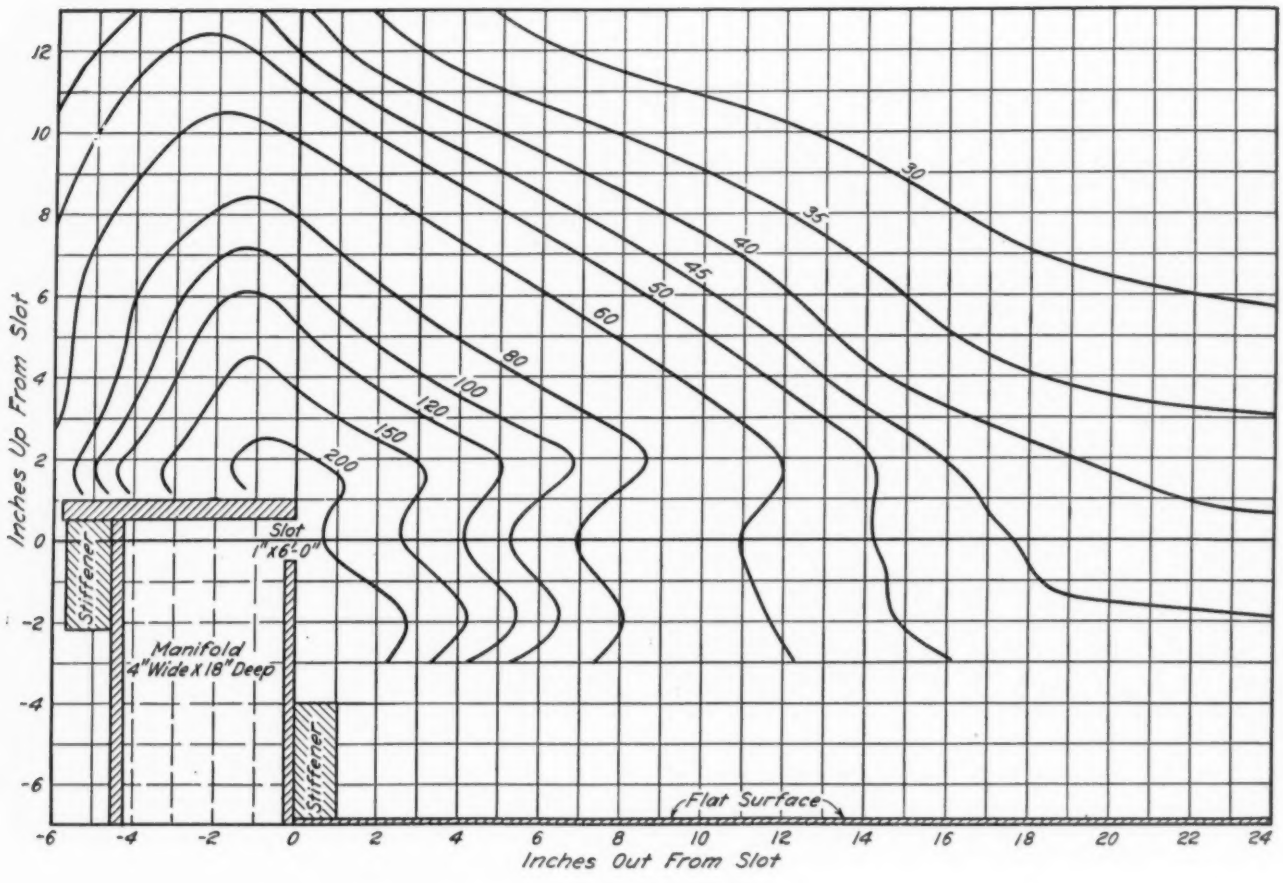

FIG. 1.
Velocity contours for plating tank exhaust slot

for the many different process tanks encountered in industry.)

The limited data on ventilation requirements for chromium plating tanks of different widths[1,2] are not sufficient to show the correctness of the new or earlier equations. It is to be noted, however, that the rate of ventilation and the power consumption for wide tanks are greater when determined by the proposed relationship than by the equation now in use. Thus, if a rate of ventilation of 100 cfm per sq. ft. of tank area is necessary for a tank 3 ft. wide, the exponential formula would require a rate of 160 cfm per sq. ft. for a tank 4 ft. in width. In the interests of economy in design, therefore, further study of the ventilation requirements on plating tanks is needed, particularly with reference to the value of the constant *K*.

Design of Exhaust Manifold

THE need for a uniform rate of ventilation through the entire length of the exhaust slot has been pointed out. This is most nearly secured by using a slotted plenum chamber of relatively great cross-sectional area in which the negative pressure is substantially uniform regardless of the location of the fan connection. Another method is to provide numerous pipe connections from the fan to equally spaced points along a slotted duct. Neither scheme is practical because of the limited space generally available around plating tanks. A tapered duct with a slot of uniform width with the fan connected to the larger end is found in practice to give a non-uniform distribution of flow. The use of an adjustable tapered slot is objectionable because the slot opening is seldom properly adjusted or fixed permanently in the correct position.

The problem becomes one of designing a compact manifold of simple construction which will provide a reasonably uniform distribution of flow and does not occupy too much space. The simplest design is one which employs a manifold of uni-

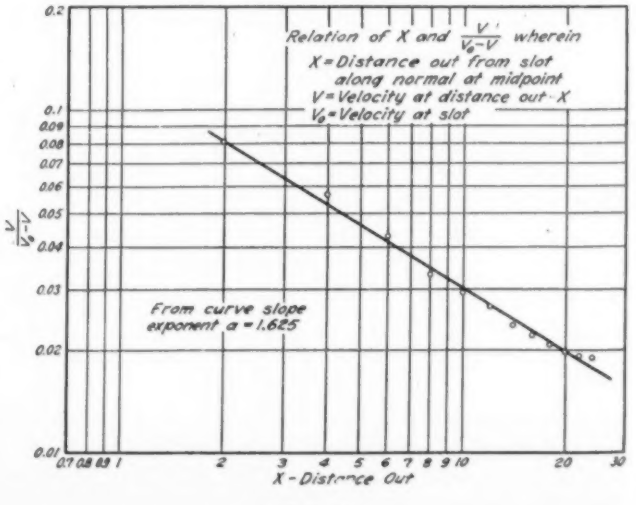
Relation of X and V'/(V0-V) wherein
X=Distance out from slot along normal at midpoint
V=Velocity at distance out X
V0=Velocity at slot
From curve slope exponent a=1.625
X-Distance Out

FIG. 2.
Relation between center line velocity and distance outward from slot

form cross-section on the two long sides of the tank with a common exhaust chamber and fan connection at one end. The cross-sectional dimensions of the manifold should be as large as possible in order to approach the effect of a plenum chamber. In accordance with common practice in ventilation, the manifold may be provided with internal vanes, as required, to insure a uniform distribution of flow. The design requirements of such a manifold are developed below.

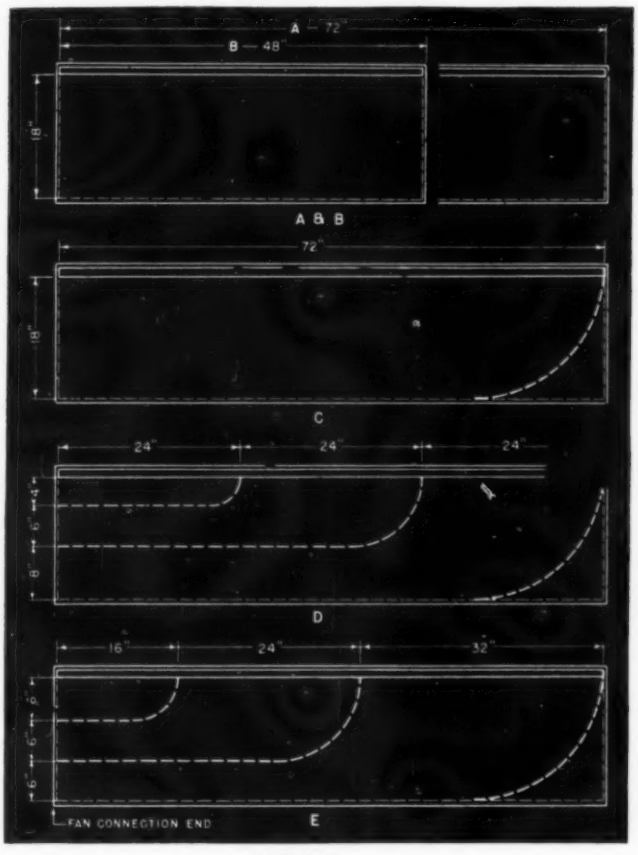

FIG. 3.
Side views of test manifolds

The test apparatus was fabricated of one-quarter inch plywood and one-half inch lumber with stiffening members, as required. Internal dimensions of the manifold were 72 in. long, 18 in. deep and 4 in. wide. A one-inch slot ran the full length of the manifold at the top of one side, thus providing an air intake area of 0.5 sq. ft. A plywood box 30 in. long, 27 in. high and 12 in. wide was joined at right angles to the manifold to serve as an exhaust plenum chamber. The fan was connected to this plenum at the rear top corner diagonally remote from the manifold with common 8 in. pipe. The additional height of plenum over that of the manifold served to simulate the air flow effect of the tapered elbow ordinarily used in practice to connect the fan pipe to the two manifolds.

A blast gate on the exhaust fan was adjusted throughout the tests so that the average air velocity through the slot was 2000 fpm.

Velocities at the slot were measured with a direct reading air velocity meter using the appropriate attachment. Readings were made at the top, middle and bottom of the slot for each one-inch interval along the slot. These were averaged to obtain one set of readings for the one-inch slot opening.

The first series of velocity readings were obtained with the simple manifold (Fig. 3*A*). The results

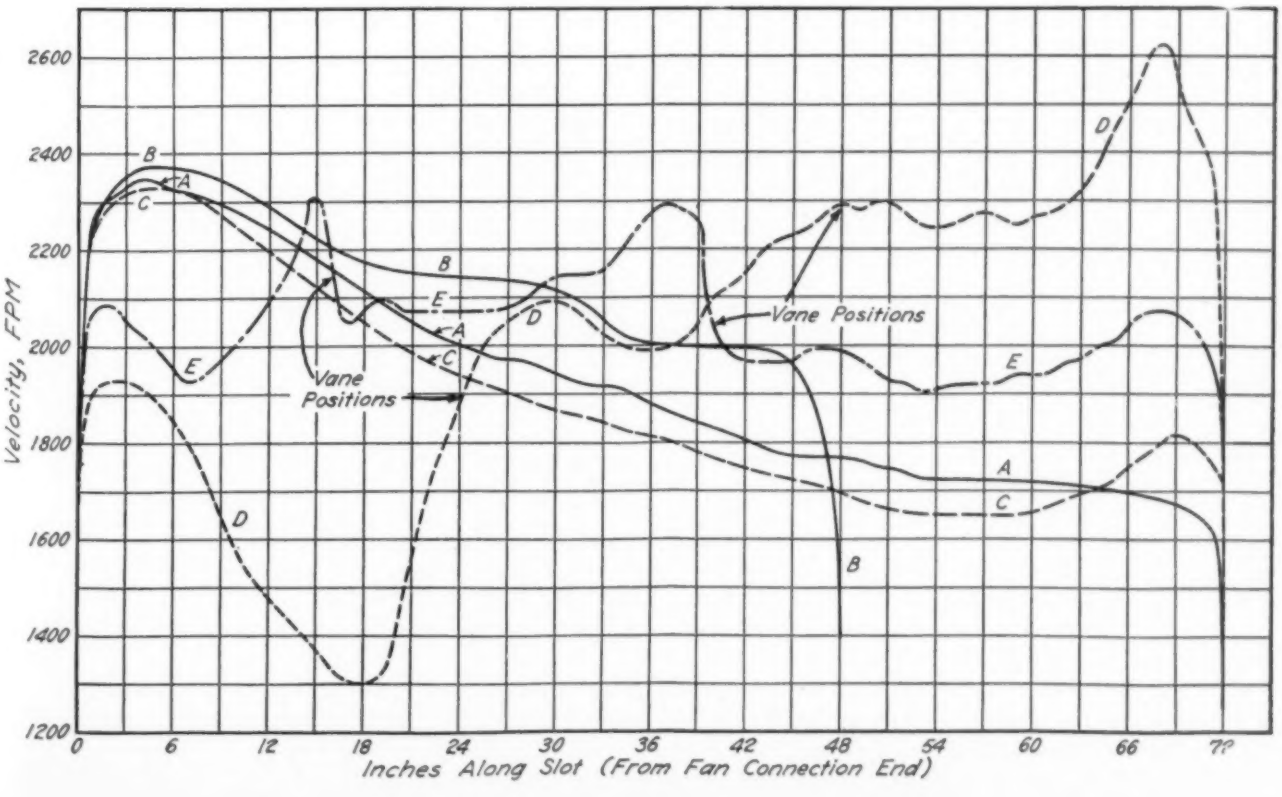

FIG. 4.
Velocity distribution along slot for the various test manifolds

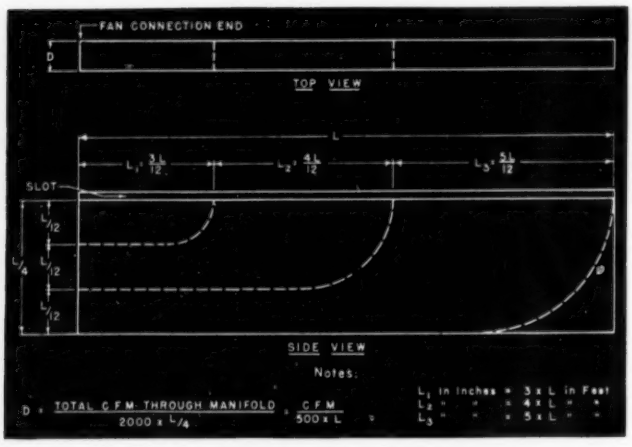

FIG. 5.
Recommended design of exhaust manifolds for uniform distribution of flow

are plotted as Curve *A* in Fig. 4 and reveal a marked decrease in slot velocity with distance from the exhaust end of the duct. In the second test, the manifold was shortened to 4 ft., thus making its cross-sectional dimensions relatively greater and more nearly approaching the plenum chamber effect (Fig. 3*B*). The velocity distribution, plotted as Curve *B* in Fig. 4, also shows a marked non-uniformity along the slot. The results of these two tests are in agreement with past experience and indicate that a simple uniform manifold of the limited dimensions permitted in practice is not acceptable. A single curved vane at the remote end of the manifold (Fig. 3*C*) served to increase the flow in this region, as shown in Curve *C* in Fig. 4, but did not significantly improve the distribution of flow through the slot as a whole. The results of this test indicate clearly the need for additional distributing vanes.

Accordingly, two more curved partitions were installed in such a way as to divide the manifold into three separate sections, as shown in Fig. 3*D* and 3*E*. In Arrangement *D*, the vanes were equally spaced along the slot and arbitrarily located vertically so as to provide the greatest cross-sectional area in the bottom section which draws air from the remote end. The resulting velocity distribution, shown by Curve *D* in Fig. 4, indicates that too much air was drawn through the bottom section. Consequently, the vanes were relocated, as in Fig. 3*E*, to provide equal vertical spacing and a varying distribution along the slot according to simple fractional proportions. With the exception of minor disturbances in flow in close proximity to the vanes, this arrangement provides a reasonably uniform slot velocity (Curve *E*, Fig. 4). Undoubtedly, further investigation would show more desirable vane arrangements, but the practical features — and particularly the simplicity — of arrangement *E* recommends it. The final design, with the proportions adjusted to require only the simplest design calculations, is shown in Fig. 5. The arrangement shown in Fig. 5 may be employed for manifolds of any reasonable length, provided all dimensions are determined in the proportions shown. For long tanks this requires considerable depth to the manifold. A more compact arrangement for long tanks is given by installing fan connections at both ends of the tank thus making the effective manifold length for each fan one-half the tank length.

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News and Notes

—of Association Activities—

THE writer finds that he lacks information concerning some of the sectional activities, and has been unable to fill in this information, owing to a change in the publication policy of this Journal.

However, the ever-active New York group held a very successful meeting January 15, 1941, at the District Health Center Building in New York, with DR. ALEXANDER O. GETTLER, Professor of Toxicology and Chemistry, New York University, as the principal speaker. DR. GETTLER's topic was "Micro-analytical Methods in Chemistry," and he explained the use of these methods in connection with the New York City work in which several thousand bodies are examined in cases of suspicious and sudden death. In the course of his talk, he described micro methods for the distilling of volatile liquids from brain matter and the determination of carbon monoxide, mercury and fluorides. He also outlined the micro methods used in his laboratory for the fractional distillation of volatile solvents and determinations of molecular weights and boiling points of liquids. At the next meeting, in the Director's Room of the McGraw-Hill Building, March 5, 1941, DR. LYDIA G. GIBERSON spoke on the subject of "Psychiatric Aspects of Industrial Hygiene."

The Chicago Section had MR. B. F. POSTMAN, Industrial Hygiene Engineer in the Connecticut State Department of Health, as a speaker on January 30, 1941. The subject "Engineering Measures for the Control of Industrial Health Hazards" was completely illustrated with lantern slides and provided a very interesting outline of various actual installations of preventive measures.

The Chicago Section heard the writer, on March 27, 1941, discuss "Control Measures as Found in Large Industry."

The Michigan Section combined with the symposium which was held at the University of Michigan in Ann Arbor January 23, 1941, to have an evening meeting. This plan, which we believe merits consideration by other sections, involves having one or two meetings a year in some other city than the one in which the majority of the members reside. DR. C. P. MCCORD gave a very interesting and frank discussion of "The Role of Industrial Hygiene in National Defense."

The next meeting of the Michigan Section, held Thursday, March 20, 1941, was different in that it was not a dinner meeting. However, the topic was about the same, with the subject matter entirely different. DR. CLARENCE SELBY discussed "The Role of Industrial Hygiene in National Defense" as he sees it from his position as Chairman of the Sub-committee acting as an advisory body on health matters pertaining to industry.

The writer would be interested in securing the views of members concerning their cooperation in sending in items of industrial hygiene importance for inclusion in this column.. —GORDON C. HARROLD, *Secretary.*

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